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THE TREATMENT OF ASTHMA.[1]

By CHARLES SUTHERLAND, M.B., B.S. (Melb.), M.R.C.P. (London),
Physician in Charge of Asthma Clinic, Alfred Hospital, Melbourne.
[*From the Baker Medical Research Institute.*]

DURING recent years our methods of treating asthma and hay fever have been improved in many ways and in this paper I shall briefly outline some of those methods which are especially useful. Formerly little hope of improvement could be given to the patient and as a rule he was condemned to put up with his attacks and to get what relief he could from inhalations and medicines. Now it is possible with careful investigation and treatment to relieve the great majority of sufferers.

In rather more than half the cases the illness seems to be due to hypersensitiveness to some substance. Of the remainder focal sepsis seems to account for many and in a proportion both these factors operate. In a few cases neither focal sepsis nor hypersensitiveness seems to be present.

In hypersensitive patients the mechanism of symptoms appears to be somewhat as follows. The exciting substance is usually present in the atmosphere and when this is inhaled and comes in contact with the hypersensitive tissues of the nose and bronchi, it liberates something which is apparently indistinguishable from histamine. The outstanding effects of histamine are spasm of the smooth muscle of the bronchioles and congestion and swelling of the mucous membrane. In large doses it will reproduce all the symptoms of surgical shock with profound fall in blood pressure.

Considered from a theoretical point of view the objects of treatment are: (i) to prevent the formation of this "histamine-like" substance by avoiding the exciting cause or lessening the sensitiveness or (ii) if this is impossible, we must combat the spasm of the bronchial muscles or the swelling of mucous membranes caused by it.

In practice circumstances will determine whether we are able to control the cause in this way or whether we must retreat on the somewhat feeble expedient of treating symptoms.

In a typical case the treatment is as follows: First try to find out whether the patient is hypersensitive to any substance. This is discovered by taking a careful history and applying skin tests with all the likely substances which are known to cause attacks. These exciting substances are usually called "atopens" or "allergens." The history in these cases is always very helpful.

Locality is often of great importance to the patient. He may be free of attacks at Mildura and constantly wheezing when in Melbourne. Some cannot live near the sea and some find that one particular house or even a particular room affects them. I have one patient who cannot go into a certain building in Collins Street without feeling an immediate tightening in his chest. Dust extract from this building gives a large reaction when applied to a scratch on his skin, whereas dust from his home gives practically no reaction.

Seasonal incidence is another very suggestive fact. If the attacks occur only in the spring time, we immediately think of pollens as a cause. Usually these patients are worse in dry, dusty weather and north winds bring on attacks.

One should always inquire whether contact with animals brings on attacks. Horse hair, cat hair and rabbit fur are common causes. Women sensitive to feathers or dust find that making beds or dusting upsets them. Substitution of a kapok pillow for a feather one often gives dramatic relief

Food is sometimes a cause and egg is probably the commonest offender. Next come nuts, milk, oatmeal, wheatmeal, pork and so on. Frequently urticaria or eczema is associated with the asthma.

Many of these hypersensitive patients get prolonged attacks of sneezing before the asthma. Their eyes and throat itch and the nose runs with clear glycerine-like mucus.

Another important diagnostic point in history is that adrenalin completely stops the attacks. If it does not, one suspects bronchitis or other causes for the dyspnœa.

Then again, if there is a family history of hay fever or asthma, it is more likely that the patient will be hypersensitive.

Specific Treatment.

If the cause can be found, it is best to avoid it completely. For instance, if the patient is sensitive to feathers, it is sometimes sufficient to get rid of any feather pillows *et cetera* and to remove all traces of feathers from the house. If the cause cannot be completely avoided, then we should attempt to desensitize the patient.

To do this we inject hypodermically gradually increasing doses of an extract of the substance to which he is sensitive. It is most convenient to have four dilutions which we will call A, B, C and D (D being one thousand times stronger than A). I always use extracts prepared and standardized according to the methods of Dr. Arthur F. Coca, of New York. Suitable extracts may be obtained from the Commonwealth Serum Laboratories, Melbourne.

First we give four injections of the weakest, A, 0·1, 0·2, 0·4 and 0·8 cubic centimetres. Next give of B 0·1, 0·2, 0·4 and 0·8 cubic centimetres. Then give of C 0·1, 0·2, 0·4 and 0·8 cubic centimetres and of D 0·1, 0·2 and 0·4 cubic centimetres.

When using the weaker strengths, A and B, two or three days is a sufficient interval between injections. Then with stronger strengths it is wise to extend the interval to seven and finally to fourteen days.

If any injection causes much local reaction, such as swelling or itching or brings on an attack, it is well to give a smaller dose until tolerance is established. Never change to a new batch of extract in the middle of a course, as it is impossible to tell

[1] A post-graduate lecture delivered in Melbourne on November 14, 1927.

how a patient will react. Never give injections without having a supply of adrenalin handy and always get the patient to wait half an hour after each injection. If an attack follows an injection, give 0·3 mil (five minims) of adrenalin subcutaneously and repeat it if necessary, but constitutional reactions produced in this way are usually a sign of bad technique, as one should get sufficient warning when the dose is getting too large.

As a rule, when the dose reaches about 0·5 cubic centimetre of the D strength there is so much local reaction that the dose is not increased. Usually it is necessary to repeat this final dose every four or five weeks for some months, as the hypersensitiveness is liable to return.

With hay fever the treatment should be commenced six weeks before the symptoms are expected and then the final dose should be repeated once a month until the season ends.

Treatment of Patients Who Do Not React to Tests.

With good technique hypersensitiveness may be detected in 70% of patients and we can expect to give these considerable relief or even complete freedom from their symptoms. On the other hand, treatment of the remaining 30% who do not react, is by no means hopeless, although the indications are much less clear and therefore we must often try several different methods before a suitable one is found.

It is better to explain to the patient that his disease is one which until recently was regarded as incurable and that treatment may quite possibly be disappointing. As a rule I find that the patient is still quite keen to try to get relief and sometimes results are quite brilliant.

With this class of case we use "non-specific" methods of treatment. Briefly, we have the choice of (i) vaccines, (ii) peptone, (iii) tuberculin, (iv) other methods such as autohæmotherapy, shock protein therapy, X radiation, diathermy.

Vaccines.

Vaccines, either autogenous or stock, sometimes produce striking improvement. It is always a good sign if the site of injection gets red and swollen a few hours after injection. As a rule it is worth trying a stock vaccine first, as results are often equal to and sometimes superior to those with autogenous vaccines. The mixed anti-influenzal vaccine of the Commonwealth Serum Laboratories is a suitable one and I usually start with 0·1 cubic centimetre of A strength and finish with 0·5 cubic centimetre of B strength, giving five or six injections. Sometimes it is necessary to repeat the last dose every five or six weeks.

I have tried this vaccine in patients known to be sensitive and although symptoms ceased for a few months, results were inferior to those obtained with specific therapy.

Peptone.

Peptone is given intravenously. The best and safest is Armour's Peptone Number 2. The initial dose is 0·1 cubic centimetre of a 5% solution, with subsequent doses 0·2, 0·4, 0·8, 1, 1·2, 1·4, 1·6 cubic centimetres. I have used it in over twenty cases and have seen no alarming reactions. Some patients have benefited greatly and others manifested absolutely no effect. It is safer always to have the patient in hospital or at any rate in bed before giving injections and always have adrenalin available.

Tuberculin.

Storm van Leeuwen, of Holland, is very enthusiastic about results with tuberculin and I have seen several patients greatly relieved by it. It is a dangerous weapon and I never treat a patient without great circumspection. In every case a von Pirquet test is done to get some idea of the sensitiveness to tuberculin and three specimens of sputum are examined and the chest is examined by X rays to exclude active tuberculosis. The patient takes his temperature each night and counts his pulse.

I usually start with 0·1 cubic centimetre of one in one hundred thousand old tuberculin and finally get up to about 0·2 cubic centimetre of one in one hundred. If there is much local reaction at the site of the injection or constitutional upset, the dose is diminished. In successful cases the attacks diminish and disappear as the higher doses are reached and then the dose is repeated perhaps every four to five weeks.

Other Methods of Treatment.

I have not had much experience of other methods, but Scott, of the London Hospital, states that he gets good results by radiating the whole body with an X ray tube placed at some distance from the patient.

Other methods, such as "protein-shock" therapy, autohæmotherapy and diathermy, seem to succeed in isolated cases.

It seems likely that there is great hope of improving our methods of non-specific treatment. It is common to find patients who have been quite free of symptoms since having had an acute illness such as pneumonia or typhoid fever. I have one patient who is always free of symptoms for three days after an attack of malaria. Then, again, some women lose all symptoms when pregnant. One patient who had suffered for years with asthma, developed myxœdema and then ceased to have asthma. When she was treated with thyreoid extract the asthma returned.

Drugs.

Adrenalin is still the best remedy. A dose of 0·18 mil (three minims) given subcutaneously early in an attack will usually stop it. If it does not, repeat it at intervals of about ten minutes until it does. It is much more effective if given intramuscularly and often in severe cases it is good to give "Pituitrin" at the same time. Stimulation of the sympathetic nerves to the bronchial muscles produces relaxation of these muscles and adrenalin

imitates the effect by stimulating the neuro-muscular junction. On the other hand, atropine acts by blocking the vagus nerves which, if stimulated, produce contraction of the bronchial muscles. Sometimes it is found that atropine is more effective than adrenalin and in these cases it is often found that reflex irritation (for example, from the nose) is the cause of attacks.

If it is likely that the patient is going to have attacks in spite of treatment, I usually teach him to give adrenalin subcutaneously. This seems justifiable, because there is no danger of habit formation and prolonged administration of the small doses used does not appear to be harmful. The essential is to get a small dose in very early in an attack. If it is delayed for twenty minutes, ten times the dose may not be so effectual. Of course, it is necessary to avoid veins as even small doses intravenously are extremely unpleasant and may be dangerous.

Ephedrine is an alkaloid isolated from a Chinese plant *Ma Huang*. It is a white crystalline powder and has a formula which is somewhat similar to that of adrenalin. Its pharmacological action is also very similar, but it may be taken by mouth and its effects are more lasting, although slower in appearing. Like adrenalin, it will raise blood pressure and it has been used in the treatment of Addison's disease. It also relaxes the smooth muscle in the bronchi and large doses will cause tachycardia and tremor. I have been using it for over two years and find it a most useful substitute for adrenalin in certain cases. However, it is not so potent in the very severe attacks and in some subjects it causes sweating and sleeplessness. The dose is thirty to sixty milligrammes (one-half to one grain).

Morphine is useful in very severe attacks and will sometimes act when adrenalin fails. It is best given with atropine. In desperate cases light chloroform anæsthesia may be necessary.

Between attacks iodides should always be tried. Sodium iodide is much better than the potassium salt and I prefer to give it in a simple mixture with a little liquorice extract and water. The complicated prescriptions often given almost invariably upset the digestion. Sometimes arsenic in small doses helps, especially in children.

Focal Sepsis.

It is most important to have a thorough examination of the nose in every case. Infection in the accessory sinuses seems to be a frequent and important factor in producing attacks. Polypi are common in these cases but, although they should be removed, results are usually disappointing. Reflex irritation is occasionally a factor and washing out an infected antrum or light cauterization of the septum may occasionally cause complete disappearance of symptoms. It is interesting to note that Brodie and Dixon found that stimulation of the nasal mucous membrane in animals will cause constriction of the bronchial muscle through the vagus.

Diet.

A great deal has been written about diet in asthma, but it is hard to get any definite guiding principles. Undoubtedly anything which upsets the digestion is liable to bring on attacks and therefore overeating is very harmful and the evening meal should be especially light. Milk definitely upsets certain patients, but it should not be cut out of the diet without good reason. Striking improvement, especially in children, often follows limitation of carbohydrates, especially sugar and biscuits. When definite hypersensitiveness to a food is found, it is better to avoid it altogether, but if this is not possible, we can try desensitization. This can be done by giving increasing doses subcutaneously, but it is usually much better to give a graded series of doses by mouth. For instance, to a child who is hypersensitive to milk, give one drop in broth, then six hours later two drops and so on up to a cup or more daily. With egg it is best to make it up in pill form to insure accuracy of dosage. Once tolerance is established it is very important to keep on with the food as a few weeks' abstention may allow the tolerance to disappear.

It should be noted that some patients do not give skin reactions to the offending food and the only ways to detect it are by the history and by feeding experiments. I have one patient who gets hives and asthma after eating almonds, but she gives no skin reaction to them.

General Hygiene.

General hygiene is also important. The patient should take regular exercise if possible and country life is usually better than town life. He should not be overdressed, but should just wear sufficient to keep warm. His bedroom should be as free from dust as possible and the ideal is simply a bed in an otherwise empty room which is frequently cleaned with a vacuum cleaner. Walls and picture rails should be frequently wiped down with a damp cloth. Symptoms often disappear when the patient is moved into hospital where there is less dust and it is well known that the majority of asthmatics lose their symptoms in high mountainous atmospheres where dust is scarce. Storm van Leeuwen, of Holland, has attempted to reproduce these conditions by building a small dust-proof chamber for the patient to sleep in and supplying it with specially filtered air. However, it is difficult to filter it efficiently without very expensive apparatus. We have built a similar room at the Alfred Hospital and find that patients sensitive to comparatively large particles, such as pollen grains, are completely relieved while in this chamber, but our filter is not sufficiently efficient to remove the fine particles of dust which upset some patients.

In conclusion it should be noted that not every skin reaction is of significance as a cause and disappointments occasionally follow specific treatment. Sometimes several different methods must be tried before an effective one is found. If symptoms do not improve and especially if the history is not typical, it is well to look again more carefully into the diagnosis.

VISCEROPTOSIS AND ITS TREATMENT.

By M. O'BRIEN, L.R.C.P., L.R.C.S. (Edin.),
Einasleigh, North Queensland.

I HAVE read with much interest Dr. Renfrew White's paper on "Visceroptosis in Relation to Posture" reported in the issue of September 10 of THE MEDICAL JOURNAL OF AUSTRALIA. I would like to make a few criticisms. He states:

> Visceroptosis is due to the combination and interaction of two causes: (i) congenital looseness of attachment of the abdominal viscera, (ii) life-long habits of defective body use.

By the latter he means the habitual assumption of the relaxed or passive posture, which he describes as follows:

> The spinal curves become marked, ... the chest descends into a position approaching that of full expiration, whilst the relaxation of the abdominal muscles together with the low position of the chest and diaphragm result in a relative descent of the abdominal viscera with a change in the external configuration of the abdomen, the lower part of which becomes very much more prominent than the upper.

His treatment consists in educating the individual, so that he habituates himself to maintaing the alert, erect figure in contradistinction to the drooped, relaxed one.

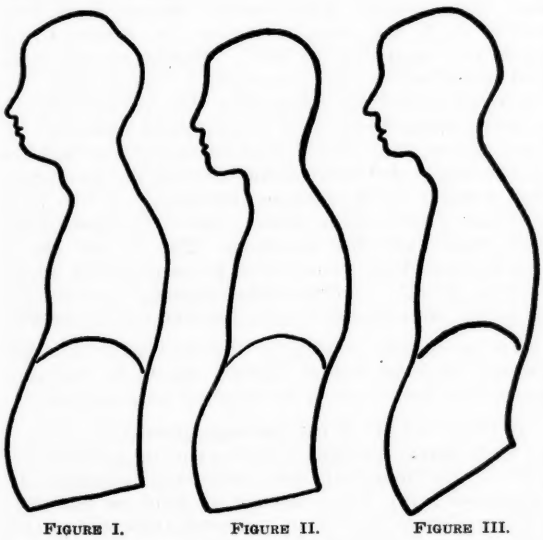

FIGURE I. FIGURE II. FIGURE III.

I have given this matter a fair amount of thought and I think Dr. Renfrew White has missed the main cause of the trouble. It is due, I am convinced, much more to a faulty build of body than to faulty habits of bodily posture. To quote Dr. Renfrew White again:

> These conditions tend to occur most frequently and in their most extreme form in that type of human being destined to a tall, slender body build.

Correct undoubtedly, for the very reason that the tall, slender man has a weakly built body.

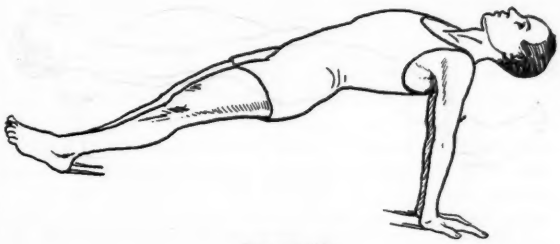

FIGURE IV.

Take the individuals shown in Figures II, III, and IV of his article and reproduced here as Figures I, II and III.

A little study ought to make clear to us the reasons of their disabilities. The principal point about their structure is that the vertebral column is weak; hence the dorsal kyphosis. The trunk walls (ribs intercostal and abdominal muscles) are also weak. The latter defect is to a great extent a consequence of the former; nature is able to adapt itself to circumstances to some extent and does not

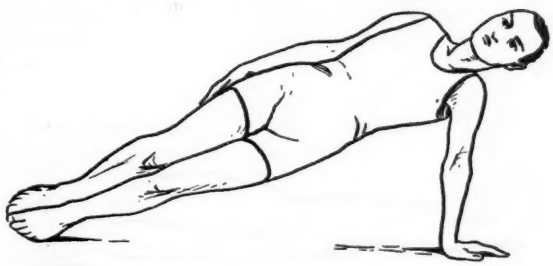

FIGURE V.

grow strong trunk walls on a weak spine. The consequences of these defects can be easily understood. Kyphosis has been mentioned. Lordosis soon follows it owing to the lower part of the dorsal spine being thrown backwards to compensate. Then the weak dorsal spine and thoracic walls cannot afford efficient support to the thoracic viscera and diaphragm which tend to descend, pressing down the abdominal viscera. Moreover, the weak abdominal muscles not affording sufficient support, the abdominal viscera descends still further, leading to a greater or less amount of visceroptosis.

Quoting Dr. Renfrew White again:

> The chest descends into a position approaching that of full expiration.

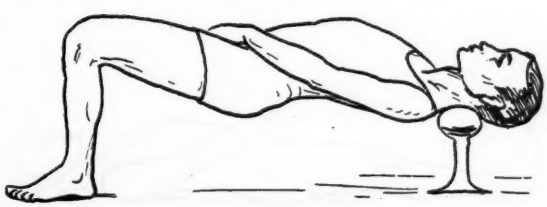

FIGURE VI.

FIGURE VII.

I think there cannot be a doubt but that this is due to weakness of the thoracic walls, especially of their musculature. When muscles are habitually given work beyond their strength, they often get tired and relax. It ought to be obvious that the descent of the chest is due to this and not to any faulty habits of posture. The same reasoning applies to the abdominal muscles.

Therapeutics, therefore, are simple. First, strengthen the vertebral column by physical exercises. Secondly, strengthen the thoracic and abdominal muscles by the same means. I have

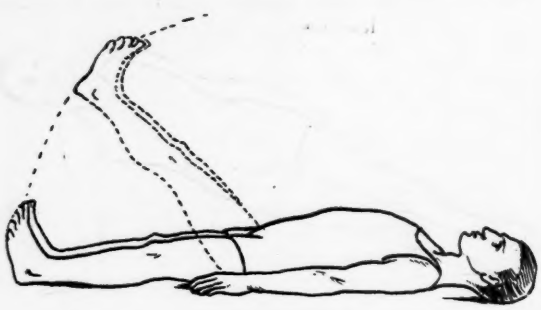

FIGURE VIII.

evolved series of exercises which are well adapted for this purpose. They are illustrated in Figures IV, V, VI and VII.

It will be easily seen that these exercises give an immense amount of work to the spinal muscles and will cause in time a great increase in their strength. An increase in the strength of the osseous structure of the spine will follow, if the exercises are taken during the growing period of life. Once the increase in the strength of the vertebral column

FIGURE IX.

is established, an increase in the strength of the trunk walls will follow automatically in the great majority of cases. If it does not, the exercises shown in Figures VIII and IX will soon bring it on.

I can assure Dr. Renfrew White that if he tries these exercises on his patients, he will find the postural treatment quite unnecessary. I have tried them on some of my patients and all of them after a little time have carried themselves in the alert, erect posture habitually and apparently without effort. Moreover, their effect in preventing visceroptosis will be incomparably greater than any possible postural treatment.

THE DIAGNOSTIC VALUE OF X RAYS IN RHINOLOGY.[1]

By C. M. EADIE, M.B., B.S. (Melb.),
Medical Officer for Diseases of the Ear, Nose and Throat, Melbourne Hospital, Melbourne.

IN opening this discussion from the rhinological side, on the diagnostic value of X rays in rhinology, I wish to express my appreciation of the honour thus conferred upon me.

I am confining my remarks to some two hundred and fifty patients whose nasal sinuses were submitted to X ray examination by Dr. Stuart Cross since the beginning of 1925. Eighty-five of these had maxillary antrum operations. In seventy the radical Caldwell Luc operation was performed, the antrum being cleared of pathological mucosa. In only one of these did I find on operation that the X ray report was misleading, in that the membrane was normal in appearance, excepting on the floor and lower part of the naso-antral wall, where there was some catarrhal swelling. The X ray report had indicated the presence of gross polypoid hypertrophy. In all the others either mucosal hypertrophy or polypi were found as diagnosed by the X ray film.

The presence of pus in the maxillary antrum, frontal and sphenoidal sinuses shown in the skiagram has been proved by clinical observation.

A report of the X ray findings gives a description of each sinus, whether it is normal or pathological and if pathological, the following changes are diagnosed: (i) The presence of fluid or mucopus, (ii) moderate degree of mucosal thickening, (iii) gross mucosal hypertrophy, (iv) presence of polypi.

Maxillary Antrum.

Moderate mucosal hypertrophy in the maxillary antrum usually denotes an early infection of low grade virulence. Polypi are clearly shown to be present and in one or two cases have been seen to disappear under treatment, establishing free ventilation and drainage. Acute inflammatory mucosa appears thickened and the difference between this

[1] Read at a meeting of the Rhinological and Radiological Sections of the Victorian Branch of the British Medical Association on May 26, 1927.

and chronic mucosal hypertrophy of moderate amount cannot always be shown on the X ray film. Empyemata can clearly be shown to be present.

The progress of the inflammatory condition of a sinus can be followed by taking X ray films at intervals of two or three weeks. It takes about three days for the fluid after a proof puncture and lavage to disappear as shown in the film, that is, in a normal antrum. A proof puncture in an antrum with gross mucosal hypertrophy may yield no fluid.

Dental cysts in relation to the antra are clearly shown and partitions are revealed.

The Ethmoid.

The fine bony walls of the cells of the ethmoid are revealed and stand out beautifully in the stereoscopic films. These walls are often shown in pathological conditions to be absent.

The lining mucosa when swollen is shown as a thickening of these septa or if fine polypi are present (not always seen on external examination of the middle meatus), then these cells appear completely clouded.

I am waiting to see an empyema of a cell demonstrated in an X ray film.

The anatomical position of the cells are well shown and an X ray film is essential if you wish to deal effectively with these sinuses.

Frontal Sinus.

The anatomical distribution of the galleries of the frontal sinus are clearly seen; this is necessary before any operative interference is undertaken.

General thickening of the mucosa is shown and in one instance what appeared to be a polypus lying on the floor was well shown in an X ray film. This, it is interesting to note, disappeared after free ventilation and drainage had been established.

The Sphenoid.

The skiagram reveals both sinuses clearly. It shows the position of the septum; empyemata and mucosal changes are demonstrated when these are present.

Conclusion.

I feel that an X ray film and full report on the nasal sinuses are of the utmost importance in diagnosis. They are essential in forming an opinion as to the best mode of treatment whether operative or non-operative.

In planning the extent of operative interference required to procure the best results, they are of the greatest value.

I will be keenly interested to learn the views held by my colleagues.

Acknowledgement.

I wish to express my admiration of the clearness of films taken by Dr. Cross and to say that I have learned to place great confidence in an X ray diagnosis of the nasal sinus condition.

THE ANÆMIAS FROM THE MODERN MEDICAL ASPECT.[1]

By A. HOLMES À COURT, M.D. (Syd.), M.R.C.P. (Lond.),
Honorary Physician, Sydney Hospital.

THE modern aspect of the anæmias is a subject peculiarly appropriate for discussion by the Sections of Medicine and Pathology, as pathological investigation plays an important part in confirming the clinical diagnosis of diseases characterized by altered conditions of the blood.

SECONDARY ANÆMIAS.

Of the secondary anæmias there is little new to record, beyond the improvement in treatment resulting from the newer knowledge of nutrition and diet, therapeutic measures being primarily governed by recognition of the ætiological factors in any specific case.

CHLOROSIS.

Of chlorosis or simple primary anæmia little need be said, except to remark that the incidence of the disease in this country at any rate is apparently markedly lessened, possibly a result of freer conditions of life, abundant sunlight, liberal diet, rational dress and generally improved living conditions.

ADDISONIAN ANÆMIA.

Of pernicious, Addisonian or hæmolytic anæmia, the case is otherwise. The incidence of the disease is apparently as great as, if not greater, than formerly and early diagnosis is more than ever important, because modern treatment holds out prospects of success previously unattainable.

With regard to nomenclature, it is possible that most clinicians are in accord with Hurst, who remarks that there is little "pernicious" about the disease except our ignorance of it and that to describe the condition as "Addisonian" or "hæmolytic" anæmia is preferable, in that these names are more rational and do not carry the implication of inevitable disaster to the mind of the patient. However, the old terminology persists.

Ætiology.

Although the recent advances in the treatment of Addisonian anæmia have wrought vast improvement in the management of the disease, it cannot be said that knowledge of the ætiology is yet on an entirely satisfactory basis.

The relationship of achlorhydria, streptococcal glossitis, infection of the alimentary tract, degeneration of the central nervous system and dietetic deficiency in the genesis of the disease is still far from complete elucidation. Evidence is, however, rapidly accumulating that the condition may ultimately be classified amongst the deficiency diseases. On the other hand, Piney[(1)] contends that a constitutional predisposition to the disease is the essential factor and that intravascular islets of megaloblastic tissue are constantly found in the

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on November 24, 1927.

hepatic capillaries, in an arrangement identical with that seen in the embryo; moreover, that pernicious anæmia can occur only in such persons as have a remnant of megaloblastic tissue in the liver which in the normal individual has undergone atrophy. He therefore regards the disease as a pathological process, engrafted on a congenital malformation. Although this view has not received universal acceptance, it appears to offer at least a rational explanation of the often observed familial incidence of the disease and accounts satisfactorliy for the occurrence of megaloblasts in the blood stream. Whether this hypothesis gain acceptance or not, there can be little doubt that blood conditions classified as pernicious anæmia may appear in association with varied morbid processes other than that recognized clinically as Addison's anæmia. Amongst these conditions are included infestation by *Bothriocephalus latus*, gastric cancer, syphilis and other infective processes, hæmolytic anæmia of pregnancy and others. Further, the apparent similarity to deficiency disease of the sprue type is a striking fact. In the present state of knowledge of the ætiology, therefore, it may be stated that what is commonly known as "pernicious" anæmia is a morbid condition of the blood which may arise as a result of various *noci* stimuli affecting the hæmatopoietic system. In the words of Gulland[2] "pernicious anæmia is to be regarded as a symptom complex rather than a disease."

A review of the pathological processes of hæmolytic anæmia would be incomplete without reference to the remarkable observations of Macht,[3] who has carried out a phyto-pharmacological examination of blood serum from cases of pernicious anæmia by observation of the effect of serum on plant growth. Macht demonstrated that whereas the growth of seedlings of *Lupinus albus* was not inhibited by serum from normal blood and from the blood of patients suffering from other diseases characterized by altered blood states, the serum of pernicious anæmia constantly exhibited a definite phytotoxic property, inhibiting plant growth. He therefore postulated a toxin as the ætiological factor in this disease. On this basis a diagnostic test has been proposed which might be usefully employed to distinguish cases of sprue and other diseases associated with a blood picture simulating Addison's anæmia.

Macht further demonstrated that irradiation of the serum of pernicious anæmia with ultra-violet rays in quartz containers rendered it less toxic and that the effect of irradiation might be further increased in laboratory experiments by the addition of certain phytodynamic sensitizers such as eosin.

Discussion of the minutiæ of blood pathology must, however, be left to our pathological *confrères* and I propose briefly to discuss diagnosis and the modern treatment of pernicious anæmia.

Diagnosis.

Early recognition of the disease is to be achieved only if it be borne in mind that diverse symptoms are to be observed long before a definite anæmia is manifest.

Of these, paræsthesia and numbness of fingers and toes due to postspinal sclerosis may be amongst the earliest symptoms, yet often regarded as neuritis, intermittent claudication or other vasomotor or trophic disturbance; weakness, tiredness and an indescribable sense of weight in the lower limbs are frequently found to be common early symptoms.

Of particular interest are cases in which nervous manifestations precede the anæmia, a variable period elapsing before blood examination gives confirmation to the diagnosis. The demonstration of achlorhydria in the presence of minor nervous symptoms should serve to affirm the clinical diagnosis before the blood picture has become typical.

Treatment.

On general principles elimination of sepsis or treatment of foci of septic absorption is clearly indicated in cases in which gross infection can be recognized.

Of the many forms of medicinal treatment which have been in vogue since Addison first described the disease in 1855, arsenic has held pride of place. Treatment of the associated achlorhydria by large doses of dilute hydrochloric acid as advocated by Hurst[4] has undoubtedly yielded good results.

I am informed by a recent visitor that Hurst has devised a method of dispensing hydrochloric acid in soda syphons and flavoured with orange, to be taken as a drink with meals, just as one under other conditions might partake of whisky and soda at dinner! Truly an elegant method of dispensing medicine.

Blood Transfusion.

With the adaptation of blood transfusion to practical therapeutics a wave of enthusiasm greeted this procedure as a means of combating the disease. In any disease, however, the essential principles of treatment are those based on ætiological rather than symptomatic indications and as there is no evidence that the addition of healthy corpuscles attacks the cause of the disease, the results have proved on the whole disappointing.

A review of the world's literature on this subject, obtained through Nelson's Service System in 1926, fully confirmed the conclusion which had been reached from personal experiences, that "whereas transfusion of whole blood was of undoubted use in tiding over a critical emergency, there was little evidence that any enduring effect was produced or that it altered materially the course of the disease." This view was propounded in a paper read before the Branch by Dr. George Bell, Dr. A. H. Tebbutt and the writer and published in THE MEDICAL JOURNAL OF AUSTRALIA.[5]

The accompanying chart shows the course of a case in which a series of transfusions each of seven hundred cubic centimetres of whole blood was given over a period of eighteen months. There is clearly an improvement in blood counts immediately following transfusion, followed in most instances by a relapse at varying periods.

Yates and Thalhemer[6] record a case in which one hundred and thirteen transfusions were administered over a period of two years to a patient who refused all other forms of treatment, medicinal or dietetic and who subsequently died.

Dietetic Treatment.

Satisfactory evidence has been adduced by various workers that blood regeneration in anæmic states experimentally produced, was favoured by a diet rich in animal protein, together with abundance of vitamins.

Whipple and Robscheit-Robbins[7] showed convincingly that liver was a valuable article of diet in the regenerating stage of posthæmorrhagic anæmia.

Following these results, Minot and Murphy[8] evolved a diet for the treatment of pernicious anæmia. The daily diet consisted of:

120 to 240 grammes of calf's or sheep's liver.
125 grammes of red muscle meat.
400 grammes of fresh fruit.
Not less than 300 grammes of fresh vegetables, particularly spinach, asparagus, cabbage, lettuce, tomato.
Sugar used sparingly.
Starchy foods in moderation.
Not more than 240 grammes of milk.
(Excess of salt to be avoided.)

Excellent results have been recorded by various writers following this scheme of diet with or without coincident medicinal treatment.

In almost all cases the blood picture has been found to return to a condition approaching the normal, with corresponding diminution of symptoms.

Results of one hundred and five patients so treated are reported by these authors[9] to date.

Recent investigations reported by Cohn[10] and others indicate that the daily ingestion of a small quantity of a non-protein fraction of liver acts in pernicious anæmia with apparent specificity as does whole liver. The use of such an extract gives promise of a simplification of the dietetic management of the disease.

Other observers, Koessler, Maurer and Loughlin[11] have shown that a diet rich in vitamin *A* and possibly *B* and *C*, too, has been of definite value in treatment.

Murphy, Monroe and Fitz,[12] in a review of the changes in composition of blood in ten cases of pernicious anæmia treated by diet on the principles of Minot and Murphy, stated that the immediate result of the diet was to cause the delivery of new red blood cells from the bone marrow into the general circulation, as evidenced first by a prompt increase in the reticulocytes in the circulating blood. At the same time there was evidence of marked reaction in the bone marrow and a decrease of bile pigment concentration in the serum.

Coincidentally there was an increasing red cell count and hæmoglobin concentration, accompanied by a progressive growth of blood tissues as a whole, as estimated by blood volume determinations.

The morphological appearance of blood corpuscles in patients so treated became essentially normal.

The following findings illustrating the blood changes in a case of pernicious anæmia treated on these principles are reported by courtesy of Dr. Leslie Utz. Striking improvement in the blood counts is to be observed following treatment over a period of two months.

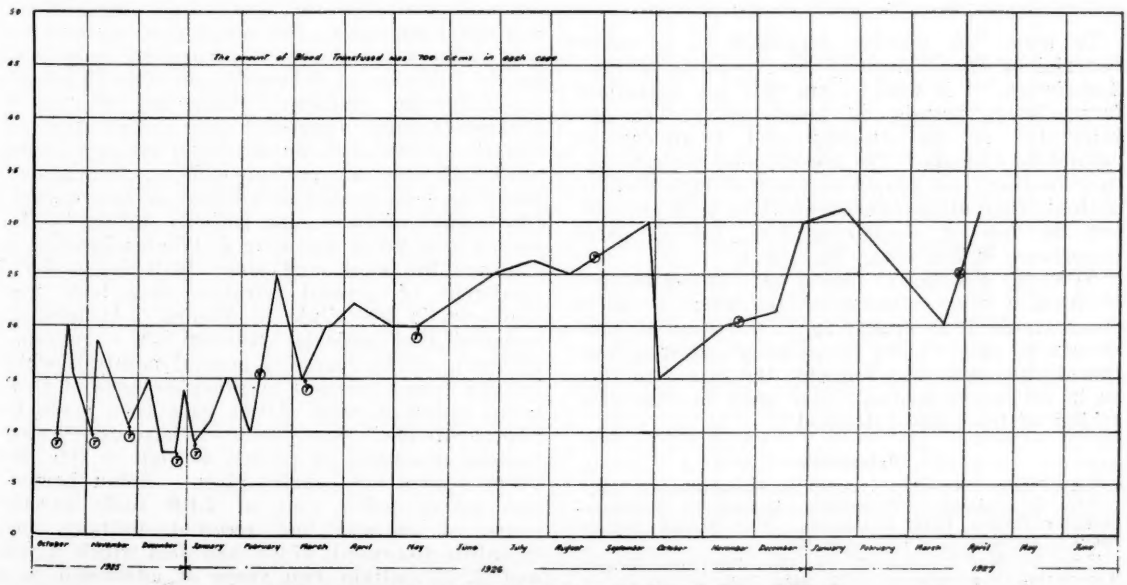

Chart of Patient Who Received Repeated Transfusions of Blood.

The blood of Mr. S., aged fifty-four years, was examined on September 13, 1927. The findings were as follows:

Erythrocytes, per cubic millimetre ..	2,400,000
Hæmoglobin value	45%
Colour index	1·07
Leucocytes, per cubic millimetre	12,800
Neutrophile cells	71%
Lymphocytes	21%
Eosinophile cells	3%
Mononuclear cells	5%

Definite leucocytosis was present. A moderate amount of poikilocytosis and of anisocytosis (megalocytosis) was present. No nucleated red cells were seen. A few cells manifested polychromasia.

The blood of the same patient was examined on October 1, 1927, eighteen days after the first examination. The findings were as follows:

Erythyrocytes, per cubic millimetre ..	3,200,000
Hæmoglobin value	65%
Colour index	1·0
Leucocytes, per cubic millimetre ..	9,400
Neutrophile cells	66%

The red cells manifested slight poikilocytosis and anisocytosis. Megalocytosis was still evident but was not comparable to that seen on the first examination. Polychromasia had practically disappeared.

At a third examination of the same patient's blood made on October 21, 1927, twenty days after the second examination, the findings were as follows:

Erythrocytes, per cubic millimetre ..	3,500,000
Hæmoglobin value	70%
Colour index	1·0
Leucocytes, per cubic millimetre	8,100
Neutrophile cells	57%

Slight poikilocytosis and slight anisocytosis (megalocytosis) were present. No polychromasia was seen.

At a fourth examination made on November 19, 1927, twenty-nine days after the third examination, the findings were as follows:

Erythrocytes, per cubic millimetre ..	4,300,000
Hæmoglobin value	76%
Colour index	0·9
Leucocytes, per cubic millimetre	9,200
Neutrophile cells	62%

The red cells were practically normal in size and shape. Neither megalocytosis nor polychromasia was seen.

To quote the opinion expressed in a recent editorial of *The Journal of the American Medical Association*,[13] it would seem that the immediate future holds promise of great advance in our knowledge of the ætiology and treatment of pernicious anæmia. The recent developments, (i) liver feeding, (ii) supply of foods of high vitamin content, (iii) ultra-violet irradiation with or without the use of sensitizing dyes, are of great importance in combating the disease.

Although a sufficient period has not yet elapsed to form a true estimate of the value of these observations, it is gratifying to feel that at least we can do rather more for patients suffering from this disease than was formerly the case. In this, as in all things medical, time must be "the true arbiter of truth and falsehood."

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THE TREATMENT OF GENERAL PARALYSIS BY MALARIA.

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THE treatment of general paralysis by malaria which has been so widely adopted in other parts of the world, has but recently been introduced into Australia and has been used in the mental hospitals of New South Wales for several months.

Improvement, rarely permanent, in mental symptoms during acute bodily illness occurs in many forms of mental disorder and is to be observed also in general paralysis. For many years pyrexia has been induced for therapeutic purposes in paretics by the injection of tuberculin or typhoid vaccine or of foreign proteins ("Phlogetan" *et cetera*). Wagner-Jauregg suggested the inoculation of malaria in 1887, but did not begin his experiments until 1917 when nine patients suffering from general paralysis were treated with benign tertian malaria. Observation of these nine patients during the following two years encouraged Wagner-Jauregg to continue his work and since 1919 the malarial treatment of general paralysis has been used regularly in his clinic in Vienna. It must be accepted that malarial therapy has a profound influence on the course of general paralysis which has not been surpassed by other methods of treatment, although great claims have been made for "Tryparsamide." Some observers have placed spontaneous remissions in paresis as high as 11%, but lower figures are usually given. Nolan Lewis[1] and others found that of 1,198 male paretics untreated or who had received ordinary antisyphilitic treatment, 47·6% had died within a year and 72·7% within two years of admission to a mental hospital. Golla[2] has summarized the

figures for the London County Mental Hospitals. Of two hundred and twenty-seven patients with general paralysis admitted during 1920 to 1923, but not given special treatment, six (2·6%) were discharged as improved within that period and 141 (62·1%) died. During a period of two years, 191 patients received malarial treatment, of whom 64 (33·5%) were discharged as improved and 40 (20%) died. Of 38 patients with a history of onset of symptoms within two to eighteen months before admission, 23 (60%) were discharged; of 25 patients with a duration of illness of nineteen to over thirty months only two (8%) were discharged. It was the general opinion of the medical officers who carried out the treatment, that those unfit for discharge were yet improved in well-being, cleanliness and general conduct. The death rate appears to have been reduced even further among a series of patients who received injections of "Salvarsan" up to a total of 2·0 grammes after malarial therapy.

Gerstmann[3] observed 400 patients after treatment in the Wagner-Jauregg clinic for two years and found that 33% had good remissions and 14% showed partial improvement.

Graham,[4] in a series of 55 patients, reported 36% considerably improved and discharged with three deaths during the treatment.

Davidson,[5] in 53 cases reported complete remissions in seven, mental and physical improvement in nine, slight mental and considerable physical improvement in seventeen, no change in five. The deaths within a year amounted to fifteen.

Lilly,[6] in 36 cases, found that ten patients were much improved and were discharged, no change occurred in fifteen, three died during and eight after treatment.

Clinical improvement may be accompanied by a reduction in the intensity of the Wassermann reaction in both blood and cerebro-spinal fluid and sometimes by absence of reaction. The colloidal gold curve frequently is changed from the paretic to the luetic type after treatment. In some cases clinical improvement occurs independently of changes in the fluids and *vice versa*.

Driver, Gammel and Karnosh[7] summarized the results in the literature of 2,336 cases of paresis in which treatment with malaria was adopted, and found that 27·5% of the patients had full and 26·5% incomplete remissions. Pilcz[8] found that of 5,000 syphilitic soldiers, 5·0% developed paresis; of the 95% who escaped, every one had had malaria, erysipelas or other infection with pyrexia within two years of contracting syphilis, while of the paretics none had a history of any pyrexial infection. These findings are not supported by experience with cases of paresis in dwellers in tropical countries, many of whom have had repeated malarial attacks since contracting syphilis.

Cases Suitable for Treatment.

It is the general opinion that the best results are obtained in cases in which treatment is adopted soon after onset of symptoms. A favourable outcome may be anticipated in the maniacal and apparently dementing varieties, also in tabo-paresis and in tabes. In the latter condition lightning pains have frequently been relieved or abolished and definite improvement has been reported in patients with severe ataxy and muscular weakness. Less favourable results may be hoped for in cases of over a year's duration, in senile patients and in the galloping form of paresis. Patients with juvenile paresis do not appear to benefit. But even those with old and deteriorated conditions have been treated with success. For instance, Grant and Silvester[9] report the case of a paretic aged fifty-two with at least a three years' history.

> His condition grew steadily worse and he had several epileptiform seizures. He rapidly lost weight, became wet and dirty in his habits and eventually developed a bedsore over the sacrum. It was then decided to inoculate him with malaria. The fever was cut short by quinine on the eleventh day owing to his exhausted physical condition and low vitality. The patient gradually improved in his physical and mental states and has now regained sufficient strength to walk about quite comfortably and without assistance. The bedsore has now completely healed. He is in a state of partial remission with a certain amount of residual psychic defect. His reaction times have improved, convulsive seizures have been absent and his speech has become more coherent and distinct.

In other bedridden, dull and confused patients mental and physical improvement have been maintained for at least a year. In those who undergo physical, but not mental improvement, there may develope symptoms of the schizophrenic type, auditory hallucinations, paranoid ideas, mannerisms and negativism. It must be admitted that malarial treatment has increased the chronic population of mental hospitals by commuting the death sentence of many paretics to one of life imprisonment.

The treatment of weak and emaciated patients is undoubtedly attended by some risk and it is seldom advisable in these to allow the malaria to run the full course of eight to twelve paroxysms. Such patients should be allowed to have four or five rises in temperature above 38·4° C. (101° F.), after which quinine should be given. Should the condition improve, another inoculation may be given later. Patients with aneurysm or with evidence of definite myocardial degeneration or with active pulmonary tuberculosis are best left alone. The presence of other grave physical disease is usually a contraindication to malarial therapy.

It will be seen, therefore, that the great majority of general paralytics and also of tabetics should be given a chance with malarial therapy.

Of the actual cause of improvement we are still in doubt. As the spirochæte is susceptible to temperatures of 40° C. (104° F.) upwards, it is possible that the therapeutic effects of malaria are due to the pyrexia. According to another theory there is a formation of antibodies after the rapid destruction of red cells. Again, we may suppose that the induced illness stimulates the general bodily reactions against the spirochætes. It may be noted in this connexion that the latter appears to be the mode of action of "Tryparsamide," which *in vitro* in much higher concentrations than obtain

in the blood after injection, fails to cause the death of the spirochæte. "Tryparsamide," like other arsenical compounds, has a tonic effect and may therefore be used in weakly patients as a preliminary to malarial therapy.

Obtaining the Blood for Inoculation.

It cannot be emphasized too strongly that the blood for inoculation should contain a strain of pure benign tertian malaria and should preferably be obtained from a source where its course and effects have been proved under skilled observation. It is not always easy to distinguish the ring forms of the benign and subtertian varieties and repeated observations are essential before a blood is selected for inoculation. Hence the danger of inoculating blood from any malarial patient newly arrived from a tropical country who may well harbour a double infection. Opinion is divided as to whether a strain gains or loses in virulence by repeated transmission through the human host. Macbride and Templeton[10] believe that the virulence becomes increased and advise that new strains should be introduced from time to time into institutions where malarial treatment is carried out. Other observations go to disprove this, for single strains have been used for prolonged periods without any evidence of increased virulence. A new strain may be required when a patient in whom reinoculation is attempted, has developed immunity to the strain with which he was first infected, a not infrequent occurrence.

When malaria is employed in the treatment of disseminated sclerosis, it is perhaps desirable that the donor should have failed to react to the Wassermann test, although there is no recorded instance of the conveyance of syphilitic infection from the blood of a paretic. Inoculation by an infected mosquito does away with possible objections, but is a less certain procedure and is full of difficulties. Moreover, Rudolf,[11] Davidson[5] and others have found that there is a greater tendency to malarial relapse after the paroxysms have been checked by the administration of quinine when mosquito inoculation has been employed.

Immediate inoculation from donor to patient is the simplest method. The blood should be drawn soon after a malarial paroxysm so that young ring forms may be present in the blood; this, however, is not of great importance. When immediate inoculation is not practicable the blood should be defibrinated by being stirred in a glass vessel with a glass rod or by being shaken with glass beads for five to ten minutes under aseptic precautions.

Should it be necessary to transmit the blood for any distance, ten or more cubic centimetres of the defibrinated blood may be sent in a tube packed round with ice in a wide-mouthed "Thermos" flask. In this way the parasites may be kept alive for at least twenty-four hours. Grant and Silverston[9] have inoculated a patient successfully with defibrinated blood kept at 0° C. for sixty-six hours.

Subcutaneous or intramuscular inoculation of about two cubic centimetres of malarial blood into three or four different sites is the method most frequently employed. Injections between the scapulæ are least likely to be interfered with by the patient. After intravenous injections the incubation period may be reduced by several days, say to within a week, but the possible introduction of clots and shock effect of foreign protein are disadvantages which outweigh any benefit, unless for any reason it is desirable to induce pyrexia with the least possible delay.

Course of Malarial Paroxysms.

The incubation period after subcutaneous or intramuscular inoculation lasts on an average from six to fourteen days; a period of thirty-six days has been recorded. After intravenous injection the incubation period is reduced to about six days. The early malarial paroxysms are rarely typical and several slight and irregular rises in temperature together with increase in pulse rate as a rule precede the more definite paroxysms, when the temperature rises mount to 39·4° to 40·5° C. (103° to 105° F.). Parasites may not appear in the peripheral circulation for two to three days after the temperature has begun to rise. In order that slight and transient rises in temperature may not be missed it is desirable to record the temperature every four hours after inoculation and every hour as long as the temperature exceeds 37·2° C. (99° F.). Once the temperature has mounted to 38·3° C. (101° F.) the pyrexia takes on a remittent character, but the typical benign tertian course may be broken by quotidian paroxysms. In fact, when the temperature is recorded at periods of two or three hours, it will be found that the typical tertian fever is the exception rather than the rule. The usual symptoms of headache, nausea, chill and general feeling of malaise and misery are rarely complained of by paretics, so that the rise in temperature and pulse rate should be watched with especial care. The patient may become restless, apprehensive and even delirious.

The blood should be examined daily for parasites after the fifth or sixth day after inoculation and during the course of paroxysms when a daily estimate should be made of the ratio between the numbers of parasites and red cells. If the patient's condition permits, he should be alowed to have eight to twelve good paroxysms with a rise of temperature to at least 38·3° C. (101° F.).

Indications for Checking Treatment.

Certain danger signals should be observed:

1. A sudden decrease in strength and listlessness and apathy between the paroxysms are important indications for checking the malaria.
2. Failure of the cardio-vascular system, decrease in intensity of heart sounds, weak pulse, fall in the systolic blood pressure to 95 millimetres between the paroxysms, hypostatic congestion.
3. Occurrence of seizures during treatment.
4. Development of any acute illness, respiratory, gastro-intestinal *et cetera*.
5. Jaundice of the skin. Less intense jaundice, apparent only in the conjunctivæ, is not usually regarded as serious, and is a common feature.

6. Rise of temperature over 41·1° C. (106° F.).

7. Sudden increase in number of parasites as shown by parasite-erythrocyte ratio. Patients vary considerably in their tolerance of infection; some may react severely when the number of parasites in the peripheral circulation is scanty. The sudden increase rather than the actual ratio is the point to be watched.

The risk of death from malaria is small when attention is paid to these danger signals. In a series of 96 cases Rudolf[12] had 29 deaths, five of which occurred before parasites or fever appeared, seven while parasites or fever were still present and 17 after the malaria had been checked. Of the 12 patients who died before the cessation of the malaria, three developed pneumonia, two died of cerebral hæmorrhage, one had pyæmia, the remaining six became progressively weaker and their deaths were attributable to malaria. One patient failed to develope pyrexia, although parasites were present in the blood. Other writers have recorded a death rate of 6% due to exhaustion from malaria.

The treatment of the patient during the paroxysms should be directed towards the relief of discomfort, but no effort should be made to reduce the temperature unless it exceeds 40·5° C. (105° F.). Moderate restlessness and delirium may be checked by veronal 0·6 gramme (ten grains). Phenacetin may be given for the relief of headache and Rudolf[12] reports good results from the oral administration of three hundred cubic centimetres (ten ounces) of 5% glucose solution every three hours. The tinctures of digitalis and strophanthus may be given where there is a threat of cardiac failure. The heart and circulation should be watched carefully throughout the treatment.

The malarial fever is terminated easily by the administration of quinine. The following routine has been found to be adequate:

Quinine bisulphate, 0·6 gramme (ten grains).

Liquor arsenicalis, 0·18 mil (three minims).

This is given twice a day for three days and then once a day for fourteen days, a total of twelve grammes (two hundred grains) of quinine. As a further precaution two daily doses may be given at intervals of a week for two or three months. The blood should be examined for parasites until several normal slides have been obtained.

Opinion is divided as to the value of following up malarial therapy with antispecifics. Reference has already been made to the results of Golla. In view of the anæmia which is so frequently produced by malaria, it is desirable to give a tonic mixture. A course of injections of "Tryparsamide" may be given with distinct benefit for its qualities as an antispecific and tonic.

Malarial therapy is best carried out in hospitals where adequate laboratory facilities exist, where the patient can remain under continuous supervision and where special rooms are fitted up with gauze in order that the spread of malaria by mosquitoes may be prevented. The difficulties of private treatment, however, can be overcome.

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Reports of Cases.

PUERPERAL TETANY, INSANITY AND ANÆMIA FOLLOWING ECLAMPSIA.

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A PRIMIPARA, *ætatis* thirty-seven, almost at term, was seen by her doctor at about 4.30 p.m. on August 18 because she suspected that labour had begun. She appeared well. Two hours later she suddenly became blind and developed an eclamptic condition. The patient was admitted to the Queen's Home in strong convulsions. Her urine became solid with albumin on boiling and her systolic blood pressure was found to be 220 millimetres of mercury and her diastolic blood pressure was 105 millimetres of mercury. The condition was diagnosed as eclampsia and the patient was isolated in a darkened room and she was given a hypodermic injection of 0·03 gramme (half a grain) of morphine followed by two grammes (thirty grains) of chloral hydrate in solution *per rectum*. Later she was given five cubic centimetres of "Veratrone." The immediate result was a lowering of the systolic blood pressure to about 165 millimetres of mercury. This indicated that the condition was not superimposed upon a previous nephritis, but that it was probably a true eclampsia due to a toxæmia (whatever that infers). Further, there was no history of renal disturbance.

Shortly after admission œdema of the feet and ankles developed rapidly. The fœtus was found to be dead, so in accordance with the Rotunda *régime*[1] no attempt was

made to deliver the patient,[2] as induction or *accouchement forcé* might only precipitate further fits.[3] Additional treatment consisted of washing out the stomach through a stomach tube; about three and a half litres (six pints) were used before the fluid returned clear. After this one hundred and twenty cubic centimetres (four fluid ounces) of *mistura sennæ composita plus* fifteen grammes (half an ounce) of magnesium sulphate were introduced into the stomach and the tube was withdrawn.

Colon lavage was next commenced and this was followed by injections of sodium bicarbonate solution owing to the probable acidosis which often occurs and also to provide more fluid for elimination.[4] The whole treatment aimed at elimination and the control of the fits.[5] Further, the patient was starved, water only being given. No venesection was made.[6]

On August 20 the patient developed strong labour pains and might have delivered herself, but for a rigid perineum. She was delivered by forceps of a macerated male fœtus. The perineum was lacerated, but before any sutures could be inserted liquid fæces were passed in great quantities, thus infecting the whole lacerated area. This was subsequently sutured after having appropriate surgical toilet. After delivery the systolic blood pressure fell from 203 millimetres of mercury to 156 millimetres of mercury. Examination of the retinæ revealed hæmorrhagic patches, but no very definite papillitis. The patient appeared to progress well for the next two days, although her urinary output was not satisfactory. At the end of two days the patient showed signs of muscular twitching followed by definite spasms. There was also considerable abdominal distension and the stomach appeared to be dilated and the dilatation was like that of acute dilatation of the stomach.[7]

At first we thought that we were dealing with puerperal eclampsia, but decided that the case was one of tetany,[8] although it seems uncertain as to whether these two conditions have not a common cause[9] due to (i) a disturbed metabolism which produces toxic substances,[10] and (ii) the associated gastro-intestinal condition.[11] Dulsace and Guillaumin found a fall of blood calcium after ovariectomy[12] and we wondered whether a lack of ovarian function had similarly produced a fall in blood calcium with resulting tetany. The spasms developed with flexion of the elbows, wrists, metacarpo-phalangeal joints. This condition is thought to be due to a reduction of the calcium ions associated with disturbance of acid base balance in plasma[13] and this is probably due in part to parathyreoid dysfunction.[14] Luckhart and Goldberg treated with calcium dogs whose parathyreoids had been removed and this prevented tetany, but during œstrus symptoms of tetany recurred.[15]

Feinberg and Lush[16] consider that tetany is frequently a sequel to eclampsia and it seems that eclampsia is a metabolic disturbance due in part to ovarian and possibly multiple glandular dysfunction and among the glands mainly affected are those of the ovarian group, namely, the parathyreoid and pancreas, the former causing unbalanced calcium metabolism.

About twenty-four hours after the onset of the tetany the patient developed mental obtuseness and finally became quite irrational, the condition being regarded as puerperal insanity, although Zangmeister[17] has reported two somewhat similar cases in which the condition was found *post mortem* to be sinus thrombosis.

The spasms called for treatment and five units of "Insulin" were given as well as some glucose. Shortly after this the blood was examined and no hyperglycæmia or acetonæmia was found. The urine did not reduce Fehling's solution and no further "Insulin" was given.[18]

The patient was now given calcium lactate and milk, the latter has a large calcium and phosphorus content.[19] Parathyreoid extract in six milligramme (one-tenth grain) doses was also given because it has been shown experimentally that when active extract is injected into dogs, it produces a rise of blood calcium.[20] In the meantime the irrational state continued, while the urinary output decreased and definite œdema occurred even to the extent of partially waterlogging the lungs. The sutures in the perineal tear were sloughy, so these were cut and the vulva was cleansed. The patient called out wildly, the eyes moved independently, roving aimlessly; the knee jerks were absent.

Bourne[21] gives puerperal sepsis as the most frequent cause of puerperal insanity, but he also includes eclampsia as a cause. In his series he found that 13·5% of his puerperal insanity cases were post-eclamptic. The patient under observation had up to this time no elevation of temperature. The blood calcium content was estimated and was found to be 8·2 milligrammes per hundred cubic centimetres of blood. This result was obtained after considerable calcium intake extending over two days.

Lumbar puncture was considered unnecessary and rather inadvisable on account of the patient's back which she had scratched and which was sloughy. Hot packs were applied to the lumbar and lower thoracic regions and because the heart showed signs of failure and because basal pulmonary œdema was present, tincture of digitalis, 0·6 mil (ten minims), was given every four hours and strychnine, one milligramme (one-sixtieth of a grain), was given by hypodermic injection every six hours.

McLean's urea concentration test showed 0·9 to be the maximum concentration. About a week after the first tetany spasm the patient improved mentally and physically. The œdema and abdominal distension were much better. The urinary output was increased and the abdomen was almost normal. The bowels acted at this time without laxatives about four times during the twenty-four hours. Usually she passed the fæces under her, so that the skin of the sacral region broke down because of pressure, œdema and the fact that it was so frequently bathed in urinary and fæcal fluid. The back was swabbed with spirits after each motion and then smeared over with compound tincture of benzoin. On the day succeeding that on which the patient became rational again, she developed the first rise in temperature to 38·2° C. (100·8° F.), with a pulse rate of 100. There was a heavy cloud of albumin and the urine was acid. Macroscopical investigation gave the impression of pus, but the catheter specimen for microscopy was lost before it could be examined. The possibility of pyelitis was considered, especially as there was pain on pressure over the right kidney. Hexamine was therefore given at once.

The temperature, however, did not swing very much and the patient manifested much pallor and there were signs of some interference with the dorsal columns of the cord. A blood count was done and the result was:

Red blood cells (per cubic millimetre) ..	1,120,000
Hæmoglobin value	3%
Colour index	1·3
Myelocytes	Present
Leucocytes (per cubic millimetre)	7,600

This indicated a profound anæmia, probably due to the toxæmia,[22] starvation and prolonged metabolic disturbance. There had certainly been an intestinal condition which may have accounted for both tetany and the anæmia to some extent, but the general metabolic upset *plus* the intestinal condition was probably the cause. Hurst[23] says that anæmia is usually due to intestinal toxæmia and that "infection cannot take place unless free hydrochloric acid is absent from the gastric contents." In this case the cause of the anæmia was possibly an intestinal toxæmia which would also produce a tetany diathesis, but there must have been some preceding toxæmia to cause the eclampsia and it is interesting to speculate whether the toxæmia was intestinal or metabolic. The anæmia appeared to be of the pernicious type and responded to treatment with liver[24] and hydrochloric acid.

The actual causation of the whole condition is in doubt, but I incline to the view that the metabolic disturbance of endocrine origin together with the added stress on intestinal organs from fœtal imposition are the main factors.

In the case under consideration the blood calcium was not sufficiently lowered to produce tetany until the removal

of the placenta, when, as pointed out by Frank and others, a good deal of ovarian hormone ceases to be produced, thus accounting for the more frequent occurrence of tetany in the puerperal phase.

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DANGEROUS HÆMORRHAGE FROM CORPUS LUTEUM IN AN APPARENTLY NORMAL OVARY.

By C. E. CORLETTE, M.D., Ch.M.,

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IT is well known that hæmorrhage into the abdominal cavity from an apparently normal ovary occurs occasionally. Serious bleeding is certainly rare, and cases of such severity as to endanger life, such as that here recorded, must be extremely rare.

Ethel G., aged thirty-eight, unmarried, was admitted to the Sydney Hospital on February 15, 1918. She had always enjoyed good health, except an attack of erysipelas of the face six months previously. She had had no previous operations. Her menses had always been regular, the period recurring every twenty-eight days, and lasting six days. During the first two days of the period she was accustomed to have left-sided pain. Between the menstrual periods there was a white discharge. Her last menstrual period began on January 17.

Her present illness began with a sudden pain referred to the rectum five hours before admission. The pain then became generalized over the abdomen, and shot up to the right shoulder. A hypodermic injection of 0·02 gramme (one-third of a grain) of morphine had been given before admission to the hospital.

On admission, she was blanched, her temperature was 36·1° C. (97° F.), her pulse was 104. There was tenderness over the lower part of the abdomen, but no rigidity. Vaginal examination revealed a mass felt behind and to the right of the uterus in Douglas's pouch.

A provisional diagnosis of ruptured ectopic pregnancy was made, and the abdomen was opened in the middle line. The abdominal incision did not bleed. On incising the peritoneum, a large blood-clot presented. The opening being made, about 1,200 cubic centimetres (two pints) of blood-clot were scooped out of the abdomen. The Fallopian tubes were examined and found to be healthy. There was no sign of ectopic gestation. The spleen and liver were next examined, but no source of bleeding could be found there. At this stage, 1,200 cubic centimetres, (two pints) of normal saline solution were injected beneath the breast. The ovaries were then again closely examined, and in the right ovary was found an opening admitting the tip of a small probe. This opening was not bleeding when examined, but it contained a little black blood, which exuded when the part was squeezed. This was evidently the source of the hæmorrhage. The spot was sewn over with catgut. The vermiform appendix was examined and found to be elongated, but it was not removed owing to the condition of the patient.

Next day, February 16, her pulse was still rapid, but her general condition was better. The temperature was 100°. She had a good deal of vomiting. Saline solution given by the rectum was not retained.

On February 18, her general condition was much better, though the temperature was 38·5° C. (101·4° F.). Her colour had improved, and the pulse was good.

The subsequent history was that of an uninterrupted convalescence, and she left hospital on March 5.

Comment.

The normal time for rupture of the Graafian follicle is said to be almost the middle of the intermenstrual period. The *corpus luteum* is undergoing degenerative changes two days before the onset of menstruation, that is, at the time when the hæmorrhage occurred. In 1918, when the case came under notice, I think it was generally believed by the best investigators(1) that ovulation took place only at the midmenstrual point of the cycle, and it seemed to me then that it must have been a hæmorrhage from a degenerating *corpus luteum*. I have discussed the question lately with several men who are disposed to interpret the case as an accident associated with ovulation. It is said that ovulation occurs occasionally at other times than at the midmenstrual point. That may be so, but while admitting that I am not able to settle the question,

I still think that in this case the balance of probability strongly favours derivation of the hæmorrhage from a *corpus luteum*.

The above clinical history was already typewritten for publication when Dr. Verbrugghen's case, recorded by him as hæmorrhage from a *corpus luteum*, appeared in the pages of this journal.[2] There is a considerable amount of similarity between Dr. Verbrugghen's case and mine. I think the diagnosis recorded is the right one, and any other explanation most improbable.

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ULCER CAUSED BY DIATHERMY.

By VICTOR DELANY, M.B., B.S. (Melbourne),
Melbourne.

MR. A. was first seen on August 15, 1927. He had a chronic ulcer involving the terminal phalanx of his middle finger. The history was that three months previously he received diathermy treatment of chilblains of the right hand. During the treatment he noticed nothing, but a few hours later two blisters appeared on the middle and ring fingers which eventually broke and a discharging ulcer followed which he had been unable to heal. As he was a tailor the condition was seriously interfering with the manipulation of his scissors.

On examination both hands were swollen and congested, somewhat acromegalic in appearance; the ulcer was quadrilateral in shape with a sloughy base and indolent, sloughy edge, the base involved the nail matrix.

He had loss of light touch as tested with a piece of cotton wool and was unable to discriminate between hot and cold test tubes in both hands as far as the wrists.

He had also a definite kyphosis of his dorsal spines with flaccid paralysis of his left lower extremity (an old standing spinal lesion). Apparently while giving diathermy treatment the operator had been unaware of his anæsthesia and had raised the temperature of the sponges so high as to cause a blister formation and subsequent ulceration.

Figure Showing Lesion on Hand.

He commenced treatment with ultra-violet light, receiving three radiations weekly. Healing was complete after fourteen exposures.

An interesting feature of the treatment was the fact that the surrounding skin did not give an erythema reaction, although long exposures were given. This was apparently due to the loss of vasodilator control to the capillaries so that the usual dilatation did not occur. Such an ulcer is comparable to the trophic variety seen in *tabes dorsalis*.

This case is reported:

1. To point out the necessity for careful examination and technique in the administration of diathermy.
2. To illustrate the bactericidal and healing powers of ultra-violet light.

Investigation disclosed the fact that the patient was suffering from syringomyelia, the large rounded massive fingers acromegalic in appearance corresponding to the condition to which Charcot gave the name, cheiromegaly. The occurrence of kyphosis in the dorsal region is not uncommon in syringomyelia.

The associated vasomotor disturbance had misled the diathermy operator into thinking that the condition of the hands was one of "chilblain" circulation.

TWO CASES OF RAPID DEATH IN ADULTS WITH ENLARGED THYMUS GLAND.

By STRATFORD SHELDON, M.B., Ch.M., B.Sc. (Sydney),
Sydney.

CASE I. F.G., thirty-eight years of age, was an engineer who had been working in a newspaper office for seventeen years. At 7.45 p.m. on June 4, 1927, after an unsuccessful attempt to move a heavy press for three quarters of an hour, he moved a few steps to get some oil when he collapsed. He lay down for a few minutes, then said he was better and would soon be "at it again." An ambulance was procured and he died on his way to hospital, being quite conscious before he died. He spoke immediately before he died, which was about 8·25 p.m.

A *post mortem* examination was made next day. With the exception of an enlarged and persistent thymus and an enlarged spleen all his organs appeared healthy. The heart was carefully examined. The muscle appeared quite normal and the coronary vessels were healthy. The thymus was estimated to weigh fifty grammes (one and a half ounces). It was not actually weighed. The spleen was quite twice the usual size and was firm in consistency.

CASE II. V. C. W., twenty-nine years of age, was a commercial traveller. At 3 p.m. on October 15, 1927, he went for a swim at Balmoral Baths. After coming out of the water he said that he felt unwell and that he had pain in his chest. He took an omnibus to North Sydney to consult a doctor and while in the waiting room he collapsed. The doctor was summoned and found him ashen grey in colour and dying. Before an ambulance was procured he died.

Post mortem examination held the next day, revealed an athletic young man, well developed. The only abnormality found was a large persistent thymus of 14·7 grammes and a large spleen of 375 grammes. (The normal weight of the spleen is 200 grammes.) The heart muscle was found to be excellent and the vessels were healthy. In both cases the brain was examined including the fourth ventricle and the *hypophysis cerebri*. I certified for the coroner in both cases that death was due to *status lymphaticus*.

In Case I a claim was made for compensation before the Workers' Compensation Commission. I expressed the opinion that the man died from *status lymphaticus* and that the strenuous work he was doing before he collapsed contributed to his death. The claim was disallowed.

The two cases are so similar that I thought them worth recording. My opinion is that both died of *status lymphaticus* and that the exertion in both cases was the precipitating cause of death.

Reviews.

MATERIA MEDICA AND THERAPEUTICS.

THE eleventh edition of "A Treatise on Materia Medica and Theapeutics" is to hand.[1]

At the outset it can be said that this is one of the finest text-books on the subject of *materia medica* and therapeutics in the English language. No drug of any value is omitted. The wealth of information on every phase of the subject is amazing.

The first volume of the first edition, compiled by the late Dr. R. Ghosh, was published in 1901. The second volume, edited by the late Sir Pardey Lukis, appeared in 1903. In subsequent editions one volume only has been published.

In the latest edition quinidine and "Insulin" receive adequate notice, the indications and contraindications for use of the former being excellently set out. The more recent non-official remedies have been given due notice, particularly thyroxin, "Bayer 205" (an effective trypanocide in sleeping sickness), "Stibosan" (an antimony preparation useful in kala-azar), other organic salts of antimony and of bismuth, mercurochrome and "Hexyl-Resorcinol." Protein therapy, serum sickness and anaphylactic shock also receive due attention. Anaphylaxis is correctly described as really a state of allergy. Treatment by serum and vaccines is very well described. A new section on radiation therapy has been added giving a short but very good account of treatment by means of ultra-violet rays and radium.

Pharmacy, dispensing of drugs, methods of administration of drugs, practical hints on prescribing are all dealt with most thoroughly and efficiently. Part IV on pharmacology is particularly good. Medical students in India are very fortunate in having so excellent a text-book and work of reference. The paragraphs dealing with drugs which cause eruptions to appear on the skin are well worth reading. It is wisely stated that "Recent observations have demonstrated that soluble lime salts administered *per os* have no appreciable effect upon the coagulation of the blood." Again, "It is a mistake to say that mercurials increase the amount of bile formed by the liver." The vexed question as to the absorption of organic and inorganic compounds of iron is well presented. As regards the treatment of syphilis by bismuth preparations, it is stated that bismuth has a more rapid therapeutic effect than mercury, but is not so quickly acting as arsenical preparations; however, the effects of bismuth are more permanent than those of arsenic. "Strontium bromide is probably the most inoffensive of all the bromide salts." The rôle of alcohol in therapeutics is very temperately discussed. It is interesting to note the observation that Hooker has proposed to use adrenalin in cases of snake-bite on account of its property of lessening the toxic effects of other drugs. The account of carbon tetrachloride and oil of chenopodium is thoroughly up to date and the chapter on vitamins is excellent. In the part on organotherapy the parathyreoids are well described and also the therapeutic employment of products of the milk and serum from goats and sheep deprived of their thyreoids such as "Rodagen," "Thyroidectin" and "Moebius's Serum." The author displays no enthusiasm concerning the employment of extracts from the thymus, liver and spleen, in fact he condemns the last mentioned as unsafe. The claims of benefit following the use of extracts of the sex glands are stated without comment.

Some criticism, however, must be offered. A few misprints occur, such as *talis* for *tales*, on page 115. Occasionally the diction is rather involved. To say that the alkalinity of the urine is increased by "all potassium, sodium, lithium and calcium salts, except the salts of ammonium," is not only badly and confusedly expressed, but is also not nearly correct. We fail also to see any reason why, as stated in the book, castor oil, copaiba and glycerin should be weighed instead of measured. The Pharmacopœia definitely gives the doses in mils and fluid drachms. In chronic plumbism the book apparently accepts the "blue line" as a definite symptom, whereas it should be stated that such line is only evidence of lead absorption and not necessarily poisoning. Potassium iodide is also recommended in the treatment of chronic lead poisoning. This is not always wise and a case is on record in which such treatment caused death from acute lead poisoning. In opium and morphine poisoning atrophine is recommended in a dose of one-twentieth of a grain repeated in a quarter or half an hour. Such heroic doses should not be given as the excessive stimulation of the respiratory centre thus caused may be followed by its exhaustion, thus adding to the perils of morphine poisoning. Digitalis is recommended in fevers, including pneumonia. Such treatment cannot be beneficial and may be dangerous, as sometimes digitalis fails to exert its special action during the period of pyrexia, but when the pyrexia subsides, the cumulative effect may be hazardous. Amongst the constituents of eucalyptus oil phellendrene is included, but the Pharmacopœia expressly excludes oils which contain much of this substance. Grindelia is not a native of the Australasian colonies. In betel chewing it is mentioned that the leaf of the betel pepper is mixed with areca and catechu. As a matter of fact it is mixed with the betel nut, which is *Areca catechu* and is totally distinct from the Pharmacopœial catechu. Reference to the use of azadiracta in medicine prompts the observation that this is identical with our own indigenous white cedar which is one of our few native deciduous trees.

[1] "A Treatise on Materia Medica and Therapeutics, Including Pharmacy, Dispensing, Pharmacology and Administration of Drugs," by the late Rakhaldas Ghosh; Eleventh Edition, by Biren dra Nath Ghosh, F.R.F.P. and S. (Glasgow); 1927. Calcutta: Hilton and Company; London: H. K. Lewis and Company, Limited. Crown 8vo., pp. 740. Price: 12s. 6d. net.

THE GASTROSCOPE AND ITS USES.

"PRACTICAL GASTROSCOPY" is the title of a work by Dr. D. Rachet, of Paris, translated into English by Dr. Imianitoff, of Brussels.[1]

The subject is first treated from an historical aspect and the reader will be surprised to find how much work has been done on a subject which must of necessity be of very little practical application.

Rachet carries out most of his observations in Dr. Densaute's clinic at the *Hôpital, Saint Antoine*, Paris. He uses a straight gastroscope, equipped with an eight volt terminal lamp, a system of lenses which focus, and an insufflation canal. The introduction of the gastroscope is facilitated by a flexible piano wire guide with a terminal knob which has been previously passed into the stomach.

The author points out that there are many contraindications to gastroscopy, and that it is of practical value only in selected cases. The instrument he passes in the left lateral position.

Rachet carried out much research in order to ascertain what advantage the field of vision of the gastroscope would gain by placing the body in various attitudes, the instrument being rigid. His object was to bring the different regions of the stomach as near as possible to the gastroscope window by placing the body in these different positions. He found that in the cadaver none of these positions would enable him to see the pathologically important pyloric region, but he discovered that he was able to see much more of this region in the more flexible live patient, placed in a position of lumbar lordosis. This attitude, he thinks, realizes the best condition for the visibility of the pylorus.

He claims that it is possible to study the physiology and living pathological changes of the stomach and it is conceivable, in our opinion, that this aspect may be more important than the diagnostic, for it is our experience that endoscopic examination of the stomach, because of the great folding and redundancy of the mucous membrane, is difficult and limited in its observative possibilities.

[1] "Practical Gastroscopy," by Jean Rachet, M.D.; Authorized Translation by Fred. F. Imianitoff, D.S.C., B.A., M.R.C.S.; 1927. London: Baillière, Tindall and Cox. Demy 8vo., pp. 158, with illustrations. Price 15s. net.

The subject may be summarized in the author's words:

> It is not yet possible to claim gastroscopy as a method of routine examination, or as the panacea for settling the diagnosis of all doubtful gastric affections. On the other hand we feel sure that this book will convince the reader of the fact that internal gastric vision is no longer a mere Utopia, but that it has at least become a daily possibility, and that this endoscopy has now passed from the realm of scientific curiosity into that of practical and fertile realization.

The book is beautifully illustrated, has many anatomical and X ray plates, as well as coloured plates and half tone sketches of the many methods of examination and research.

LISTER.

THE LISTER CENTENARY VOLUME, by A. Logan Turner, is what we would expect from the editor and the son of so distinguished a father.[1]

So many addresses have been published in all our medical journals during Lister's centenary year that it is unnecessary here to indicate much more than the scope of the volume under review. Our object is to urge that it should be in the library of every physician and surgeon and that, further, it should be carefully read. As a compilation of Lister's life, work and example it is extraordinarily exhaustive and in very small compass.

After a foreword by the editor it includes a biographical sketch by George T. Beatson who ends by suggesting that Lister's sentiments were similar to those of Charles Dickens:

> Not myself, but the truth that in life I have spoken,
> Not myself, but the seed that in life I have sown,
> Shall pass on to ages—all about me forgotten.
> Save the truth I have spoken, the things I have done.

Next comes an account of the state and the range of surgery "Before the Dawn," by Alexander Miles, who insists that the surgeons before the dawn have never been surpassed as skilled craftsmen; that especially in the pre-anæsthetic days speed, dexterity and strength of hand, were absolutely essential attributes of a great surgeon. Also that many of the surgical procedures of that time have never been improved upon. But something more was wanted than supreme technical skill and this Lister gave to surgeons and in doing so relieved those who followed him of "a suppressed uneasiness that there was something that thwarted their best efforts—they knew not what."

Edward Sharpey Schafer contributes an outline of Lister's early physiological researches which emphasized the exactness and completeness of his observations on non-striped muscle, on inflammation, on blood-clot *et cetera*.

Included are Lister's Introductory Lecture as Regius Professor of Clinical Surgery in the University of Edinburgh in 1869 on "The Causation of Putrefaction and Fermentation" and his address when opening in August, 1875, the Surgical Section of the British Medical Association, on "The Effect of the Antiseptic Treatment upon the General Salubrity of Surgical Hospitals." Each address is a model of scientific teaching given with a brevity which deserves imitation and illustrates also Lister's scrupulous care in recording what he owed to previous workers in the same or allied fields.

Thirteen pages entitled "Obiter Dicta" give short extracts from Lister's published works; amongst them this sentence is typical of him:

> I desire to correct a mistake into which I fell when investigating the fermentation of milk some years ago; for next to the promulgation of new truth, the best thing I conceive that a man can do is the recantation of published error.

John Fraser, the present Professor of Clinical Surgery in the University of Edinburgh, writes upon the influence of Lister's work, calling his contribution "The Philosophies of Listers' Genius." It may truthfully be said, he writes, that Lister:

> created anew the art of healing . . . . Into this distressing atmosphere of dark self-complacency there stepped a man whose vision pierced the veil, who saw beyond the barrier the limitless possibilities of a future brighter than could have been imagined in the wildest dream.

The questions that persistently occupied his thoughts were:

> Is there no better way? Are there no means of dissipating the shadows of infection and of death that overcast the work of our hands?

In finding answers to these questions, Lister, Fraser writes, "put surgical work on such a footing that it fulfilled in part at least the exacting definition of a science" and he put it there because of his own "extraordinary passion for facts, his desire for accuracy of observation and precision of statement. He applied to surgery this attribute of scientific doubt, of painstaking laborious observation and of ceaseless observation of facts. He inherited a handicraft" from his predecessors, "he bequeathed a science" to those who followed him.

Following are short reminiscences of "The Chief" by some of his house-surgeons, clerks and dressers, all of these eminent men who had carried the torch far and wide.

Perhaps the most convincing and complete is that of Francis Caird, only recently deceased, who followed Lister after a good many years' interval as Professor of Clinical Surgery at Edinburgh University. He was a dresser and a clerk to Lister and followed as a house-surgeon and assistant to John Chiene, upon whom, in Edinburgh, Lister's mantle fell more than on any other, when Lister was translated to London.

Our own Hamilton Russell's reminiscences of Lister from his junior studentship to and including his house surgeonship are delightful reading. He ends with this reference to Lady Lister:

> The world will never know its debt to this noble woman for her share in the laying of the foundations of modern surgery. Those, however, who are able to remember her, will know very well indeed that any account of Lister and his work would be hopelessly wanting were not a humble tribute of veneration and gratitude offered to her memory.

The volume concludes with some of Lister's letters to Pasteur, William Turner, John Chiene and others and with a reproduction from John Chiene's "Looking Backward," of a photographic group of Lister and his fellow residents, as well as a short biographical notice of each.

[1] "Joseph, Baron Lister: Centenary Volume, 1827-1927." Edited for the Lister Centenary Committee of the British Medical Association, by A. Logan Turner, M.D., LL.D. (Edinburgh); 1927. Crown 4to., pp. 197, with illustrations. Price: 10s. 6d. net.

COMPENSATION FOR INJURIES TO WORKERS.

IN "Medico-Legal Injuries"[1] Archibald McKendrick has set out to give an outline regarding accidental injuries in a simple and non-technical manner and to bridge the gulf between the non-medical and the higher technical terminology used in the ordinary medical textbooks. It is difficult to judge whether he has succeeded, but he has certainly written a book of considerable value to the medical profession. Perhaps if he had aimed at writing for the medical profession only the book would have been still better. The author has obviously had a very large experience as medical referee under the *Workmen's Compensation Act* and as consulting radiologist to the Edinburgh Royal Infirmary and he treats his subject in a somewhat dogmatic manner. There are amongst others, chapters on bone and joint injuries, back injuries, heart strain, hernia and rheumatoid arthritis, all of which are so often dealt with in Australian courts. Without reviewing these in detail we may say that McKendrick deals with all of them in a sound and sensible way. His book may be commended to all those whose work brings them into special relationship with workers' compensation, whether as practitioner, referee or assessor.

[1] "Medico-Legal Injuries," by Archibald McKendrick, F.R.C.S. (Edinburgh); 1927. London: Edward Arnold and Company. Demy 8vo., pp. 350, with illustrations. Price: 18s. net.

The Medical Journal of Australia

SATURDAY, JANUARY 7, 1928.

A Retrospect.

THE year 1928 is opening with great promise. At no time in the past have there been so many earnest workers endeavouring to throw light on the countless problems in medicine that await a solution. Each year some progress is made, some new knowledge is attained. But as more is discovered and exact information is substituted for beliefs and generalizations, fresh fields for investigation are opened and new avenues are explored. It seems as though there were no limit to the number of subjects that still demand explanation. A correspondent has recently expressed the opinion in our columns that "most of the aspects of surgery have been exhaustively considered" and that it was "difficult for a young graduate to find a field of original work that has not been already dealt with thoroughly." If this were so, it would indicate that surgery is not a science, but merely an art or a craft. It would remove surgery so far from the rest of medicine that it might be advisable to hand it back to the barbers. Others, however, do not share the opinion of our correspondent with the result that the amount of original work on problems connected with surgical pathology, diagnosis, the application of physics and chemistry to surgery and surgical treatment has increased tenfold during the past five or six years.

The Medical Profession in Australia.

The growth of the medical profession continues at a relatively uniform rate. The increment is rather more than twice that of the general population. There is, however, no acute overcrowding comparable with that in the old world. The growth of specialization and the creation of pseudo-specialties equalize some of the increment; the number of medical practitioners employed on salary by governments is increasing and the development of industrial hygiene has brought with it the employment as part-time officers of a considerable number of practitioners. There are, on the other hand, indications of a lean time in the near future. At each of the medical schools of the three large universities the number of entrants is falling off. The decrease commenced a few years ago and is greater than it was. The 1928 final year will be substantially smaller than the 1927 year at each of the schools. From all accounts it appears that the standard of efficiency is well up to the average.

The British Medical Association.

Early in the year 1927 Sir John Goodwin, K.C.B., C.M.G., D.S.O., was appointed Governor of Queensland and in June he took up his duties as representative of His Majesty. As a member of the British Medical Association he became transferred to the Queensland Branch. The importance of His Excellency's membership of the Branch has been recognized. The medico-political and scientific activities of this Branch have increased rapidly during the past few years and more particularly in 1927. The interest of members resident in towns far removed from Brisbane is being maintained by a system that has been found to be of value in other States. Meetings are held under the auspices of the medical practitioners of the district and one or two representatives of the Council journey from the metropolis. The Branch has made substantial progress in its endeavour to find a solution of the hospital question. Mention should also be made of the institution of sections for special branches of medical knowledge. In March, 1927, a surgical section was formed and the initial steps have been taken for the setting up of an obstetrical section. Proposals have been put forward for the erection of a new house for the Branch.

The New South Wales Branch has been compelled to relinquish its fine premises in Elizabeth Street. The property has been resumed by the City Council on account of a scheme for the widening of the street. The Branch at present is a tenant in the B.M.A. Building. A valuable site has been secured in Macquarie Street and a prize has been offered for the best plans for a new house. The new building promises to be handsome. It is proposed to erect a large house with rooms, flats and other apartments to be let, as well as ample accommoda-

tion for the Branch. Although the capital outlay will be considerable, the returns should be commensurate. The Branch has now a membership of over 1,600. There are eleven special sections. The medico-political activity of the Branch is still tending to increase.

The Victorian Branch has suffered a great loss during the year 1927. Richard Joseph Bull died in May, 1927, during his term of office as President of the Branch. A great deal of important work has been carried out by the Branch in the twelve months, both in connexion with scientific matters and also of a medico-political nature. As in New South Wales there have been no new developments, but a considerable amount of extension of the existing work.

In the other Branches, the South Australian, the Western Australian and the Tasmanian, the same spirit of augmented activity, of increased endeavour to fulfil the aims of the illustrious founder of the British Medical Association has been in evidence. No new machinery has been forged to attain the ends in view, but free use has been made of the old. The South Australian Branch is taking steps to erect a fine new home on a site in North Terrace.

The Universities.

While the year 1927 has not been signalized as a year of innovations in the medical schools of the Universities of Sydney, Melbourne and Adelaide, signs of impending reform are not wanting. At Sydney the preparatory work of the establishment of a school of tropical medicine and hygiene has been pressed forward and is now far advanced. The Cancer Research Committee's work has developed a stage and it has been announced that Dr. Findlay has been appointed the director. The vacancy has not been advertised in this journal. On many previous occasions we have had cause to point out that the mode of election of medical practitioners to important university positions is unsatisfactory and detrimental to the interests of Australian graduates in the Commonwealth.

At the University of Adelaide Dr. H. H. Woollard has been elected Professor of Anatomy in succession to Professor F. Wood Jones. The death of Richard Joseph Bull rendered vacant the position of Bacteriologist to the Melbourne Hospital and lecturer in bacteriology at the University of Melbourne. The need for a revision of the duties attaching to the position is evident. We have urged that it is not in the interest of medical education nor conducive to progress to compel the lecturer in bacteriology at a great university to maintain his department by remunerative diagnostic work. The Medical School of the University of Melbourne ranks among the important medical schools of the world and has the reputation for first class teaching. This school cannot maintain its position unless essential chairs of medicine, surgery, preventive medicine and bacteriology are created within a short time.

The foundations for a medical school have been laid in Brisbane. A department of anatomy has been established and facilities are provided for graduates in medicine to carry out work in anatomy.

The College of Surgeons of Australasia.

The College of Surgeons of Australasia has been definitely established. The Founders have drafted a constitution and this has been adopted at the first meeting in Dunedin in February, 1927. Subsequently Foundation Fellows have been elected in each State of Australia and in both islands in the Dominion of New Zealand. The first annual meeting of Fellows will be held in Canberra on March 31, 1928. There has been some criticism of the methods adopted by the Founders in the initial work of creating the College. Criticism of a constructive kind is always beneficial and helpful. If it is merely destructive or captious, it recoils on the critic. It is not claimed that the Founders have made no mistakes. They are human, not superhuman. Moreover, they have undertaken a task that is bound to awaken opposition and perhaps heart-burnings. It is a task best attempted by men who are either retired from active practice or who occupy senior positions in the profession. The objects have been clearly defined and must meet with the approval of all who hold in high esteem the dignity and honour of the surgeons of Australasia. The public is to be informed concerning the constitution and significance of the College of Surgeons of Australasia; its objects will be explained and its methods will be elaborated. This

will be done by Sir George Syme, the doyen of surgery of Australasia, a man now retired from practice. The article will receive the approval of the Council of the College. The public will then learn that this new institution has the self-imposed task of maintaining a high standard of surgical practice in Australasia and of raising this standard and also of demanding of its Fellows and Members a code of unimpeachable ethics. The College will leave untouched the general practitioner who is compelled on account of distance from university centres and metropolitan hospitals to undertake emergency surgery. Everyone admires the skill and resource of the men who rise to necessity and who often save life by undertaking operations that usually demand much experience and constant practice for their successful performance. It will have no jurisdiction over the general practitioner who regards himself as a surgeon, but who has undergone no special training to justify his claim. On the other hand it will embrace in its membership all the leaders in the surgical world and within a short time the only criticism will be from those who seek in vain to be admitted.

Australasian Medical Congress (British Medical Association).

The second session of the Australasian Medical Congress (British Medical Association) was held in Dunedin from February 3 to 10, 1927. The session was a triumph of admirable organization. Its success not only reflects the greatest credit on the skill, hard work and resource of the Honorary General Secretary, Professor W. P. Gowland, but proves the wisdom of entrusting the work to a chosen team of first class organizers. It would be difficult to discover a more capable combination of office bearers working under the eminent President, Sir Louis Barnett, than Professor Gowland, Professor A. M. Drennan and Dr. D. W. Carmalt Jones.

The Transactions have been published in a series of supplements to THE MEDICAL JOURNAL OF AUSTRALIA and issued together with the journal week by week to the members of the Branches of the British Medical Association both in Australia and in New Zealand. The papers and the discussions are of a high standard of excellence. The letterpress of the Transactions is scarcely less extensive than that of the first session held in Melbourne in 1923, but the illustrations have been much more numerous and more difficult and more costly to reproduce. The advantages of this method of publication of the Transactions are obvious. Under the old arrangements the Transactions appeared at least one year and often two years after the session had closed. The new scheme insures publication while the subjects are fresh and the Transactions reach six thousand readers instead of as many hundreds.

The third sessions will be held in Sydney in the second half of September, 1929. Sir Alexander MacCormick has been elected the President, Dr. A. A. Palmer and Dr. T. W. Lipscomb the Honorary Secretaries and Dr. W. H. Crago the Honorary Treasurer. An executive committee has been appointed and some preliminary work has been undertaken. Various proposals concerning the organization of sessions of Congress have been suggested by Dr. Carmalt Jones and are under consideration by the Federal Committee.

National Insurance.

During the course of the year the Branches of the British Medical Association in Australia have been invited by the Federal Government to put forward proposals in connexion with the work of certification of persons claiming compensation under the national insurance scheme. It has been determined that the insurance will not include medical benefits, but that sickness and other benefits will be extended to the whole of the community. In order that each claim may be controlled, those responsible for the administration of the scheme will need certificates from medical practitioners in which the nature of the incapacitating disability will be set forth and an estimate of the probable length of the incapacity will be given. The cost of these certificates to the Government will be very great and it has been suggested that a sum of money should be handed over to the Branches of the British Medical Association to cover the total cost of certification in one year. If this were done, the Branches would have to accept the duty of insuring that the information given in the certificates is reliable and

that unnecessary certificates are not issued. As an alternative the Branches of the British Medical Association in Australia have been asked to determine a fair remuneration of the members for the necessary certificates. That someone will watch the certificates with great care in order that medical practitioners who issue loose certificates, may be severely punished is essential. When a large amount of public money is involved, the authorities have the obligation of protecting the public against all degrees of fraud. The Federal Committee has adopted resolutions regarding the supervision of certificates and has expressed the views of the Branches concerning the nature of the certificates to be issued and the fees to be paid for them. It is anticipated that the measure will shortly be brought before Parliament.

Post-Graduate Study.

The year 1927 will stand out as a year of special importance in regard to post-graduate work in Australia. The Melbourne Permanent Committee for Post-Graduate Work is to be congratulated for having arranged with Professor Allen Kanavel and Professor Charles Elliott to visit Australia and to deliver a series of lectures in August. Both are masters of their own branches of medical science, both occupy very prominent positions in the United States of America and both have been willing to contribute to the continued education of Australian medical practitioners.

In addition to this development the post-graduate work has been extended in Victoria and also in Sydney and is now a permanent institution. Reference should also be made to the progress that has been made in this connexion in Brisbane. There are no indications that the organizers of the movement will rest on their laurels. There is room for increased activity and for more centres of post-graduate study.

Preventive Medicine.

The conception that the most important part of the work of the medical profession is to prevent disease is gradually receiving more attention and fuller recognition. The preventive aspect was emphasized throughout the proceedings of Congress and the majority of the speakers took this aspect into consideration when dealing with particular diseases. The Sections of Surgery, of Radiology and of Pathology and Bacteriology passed a resolution to the effect that hydatid disease should be recognized as a notifiable disease in Australia and that stricter attention should be paid to notification in New Zealand. It was further pointed out that steps should be taken at slaughter yards and elsewhere to prevent the spread of this disease. The Section of Preventive Medicine adopted a series of resolutions in regard to the public health nurse. It also recommended that a better control should be exercised over the sale of iodized salt in order that the incidence of goître might be held in check. The Section of Pathology and Bacteriology recommended that chairs of bacteriology to include immunology should be established at each medical school in the universities in Australia. The declared object is to further medical research, medical education and the public health. In other words it is held that the more effective teaching of bacteriology and immunology would result in the better control of disease and the wider grasp of the conception of prevention. These recommendations were forwarded to the Federal Committee and this body has taken action in connexion with each matter.

The Branches of the British Medical Association in Australia have discussed various subjects connected with preventive medicine and in many instances an endeavour has been made to further schemes for the preservation of health. In particular the Queensland Branch has put forward proposals for the combating of tuberculosis, for the development of industrial hygiene and for the control of cancer.

Industrial hygiene is becoming a practical subject and the advice and assistance of the medical officers of the Industrial Hygiene Division of the Department of Health of the Commonwealth are being sought by an increasing number of firms in all States. Much excellent work has been carried out by Dr. C. Badham in the new branch dealing with industrial hygiene of the Department of Public Health of New South Wales. Mention has been made above of the establishment of a school for tropical diseases and hygiene at the University of Sydney.

Obstetrical Practice.

Since the appointment of Dr. A. Marshall Allan, M.C., in September, 1925, as Director of Obstetrical Research in Melbourne a fresh impetus has been given to the important subject of the safety of mothers and their babies. The campaign began at a considerably earlier date, when Dr. Fourness Barrington, Dr. J. W. Dunbar Hooper and several other men with wide vision and understanding took up the cudgels on behalf of the mothers. The appointment of Dr. J. C. Windeyer as Professor of Obstetrics in the University of Sydney has effected a great improvement in the teaching of the art and science of obstetrics in Sydney. Late in 1926 Dr. Marshall Allan issued a valuable interim report on maternal mortality and morbidity in Victoria. About the same time a committee of obstetricians in Melbourne presented an important report on the standardization of obstetrical treatment. In effect this report is a critical analysis of the results of the treatment of patients attending the Women's Hospital, Melbourne, in the year ended June 30, 1926. The effect of these fearless and frank discussions of facts has been the recognition of the causes of disaster to the puerperal woman and of the maternal conditions that endanger the life of the unborn infant. It is doubtful whether we are justified in dealing with this subject under a separate caption. It is in fact a specific instance of preventive medicine.

Obituary.

Death has taken an unusually large toll of the medical profession during the year 1927. Not even during the terrible years of the war when our bravest laid down their lives for their beloved country has the medical profession suffered more numerous losses. Among the senior members of great eminence were Thomas Henry Fiaschi, William Moore, Andrew John Brady, Andrew Stewart, Edward Harold Binney. The list contains the names of younger men who had established their claim for recognition among the world's scientists, Eustace William Ferguson, Richard Joseph Bull, James Matthew Petrie, William Edward John Paradice. But these are not the only men well and honourably known to their colleagues and highly admired on account of their attainments and personal qualities, who have passed hence. The names of William Frederick Taylor, of Edward Albert Officer, of George Horne, of Charles William Reid, of Robert Lee Brown, of Robert Bernard Pearson Monson and of Ralph Aliston Whitfeld may be taken from the list without exhausting it. None of these men will be forgotten by their sorrowing contemporaries.

The Australasian Medical Publishing Company, Limited.

In a couple of months the Australasian Medical Publishing Company, Limited, will have been in possession of The Printing House for three years. The development of the printing business has been more rapid than is usual in new undertakings. In the first year a considerable amount of special work was obtained and The Printing House became well known in the trade as a house with a reputation for good work. In the second year the amount of contract work increased and the task of stabilization was begun. In all new printing businesses there is a difficulty to adjust the capacity of the typesetting department to that of the machining department. In ordinary commercial printing houses it is possible to seek work suited to the plant. In The Printing House provision had to be made for the printing of scientific periodicals and other publications and the nature of the work available necessarily determined the adjustments that had to be made in regard to staff and plant. The undertaking has now reached a stage at which the transactions and other publications of the scientific bodies in New South Wales can be produced efficiently and with dispatch. New plant has recently been installed and is now fully employed.

THE MEDICAL JOURNAL OF AUSTRALIA and the Transactions of the Australasian Medical Congress (British Medical Association) were published each week for a period of over three months. The journal did not decrease in size while the supplements were being issued. The members of the Branches have supported the journal in a loyal manner and it has thus been possible to maintain the quality of the contents of the individual issues. The good wishes of THE MEDICAL JOURNAL OF AUSTRALIA for the year 1928 are extended to its readers.

Abstracts from Current Medical Literature.

THERAPEUTICS.

Antistreptococcal Serum.

W. SANDERSON, W. H. CAPON and H. H. MACWILLIAM (*The Lancet*, July 2, 1927) report the recovery of two patients suffering from streptococcal septicæmia following treatment with concentrated scarlatinal streptococcal antitoxic serum. In one case a child, aged six and a quarter years, developed severe septicæmia following *otitis media*; hæmolytic streptococci were isolated from the blood. Three cubic centimetres of 1% solution of mercurochrome were injected intravenously and several brands of polyvalent antistreptococcal serum were used by subcutaneous injection every day for some days. Nine to twenty cubic centimetres were given every day with no relief. Ten cubic centimetres of antiscarlatinal serum were then injected daily for six days with immediate and continuous good effects; the patient recovered. The other patient, suffering from puerperal septicæmia with hæmolytic streptococci in the blood, was treated with one hundred cubic centimetres "Collosol Iodine" given intravenously, parathyreoid extract and "Kalzana" with no benefit. Antistreptococcal serum twenty cubic centimetres and antiscarlatinal serum ten cubic centimetres were then given intramuscularly on successive days and the two doses of ten cubic centimetres of concentrated streptococcal antiserum prepared from a puerperal streptococcus. On the day following the last injection the temperature fell to normal and remained down; the patient recovered rapidly. In both cases the patient was seriously ill before the concentrated serum was used.

Digitalis in Pneumonia.

F. MCPHEDRAN (*Canadian Medical Association Journal*, October, 1927) discusses the non-specific treatment of pneumonia. As a rule in pneumonia death is due to vasomotor paresis which causes a rapid rise in pulse rate and a fall in blood pressure. Toxæmia produces these effects which are comparable to those of histamine shock; they are accompanied by cyanosis. There is little evidence that heart failure due to myocardial degeneration is the cause of the cyanosis, rapid pulse and falling blood pressure. Digitalis in adequate doses, that is fifteen cubic centimetres of the British Pharmacopœia tincture per forty-five kilograms (one hundred pounds) body weight, raises the blood pressure and has a valuable clinical effect. Digitalis should be given early, since its action is slow; a patient should receive about sixteen cubic centimetres (four fluid drachms) of good British Pharmacopœia tincture in twenty-four hours. Two cases are quoted in which 1·8 mils (thirty minims) were given every three hours for ten and thirteen doses respectively with the production of heart block (complete), which lasted for three days. Recovery was recorded in both cases.

Endarteritis Obliterans.

H. VAQUEZ and J. YACOËL (*La Presse Médicale*, May 18, 1927) report the results of treatment with "Insulin" of patients suffering from non-diabetic obliterating arteritis. Four patients, aged thirty-seven to fifty-one years, were studied. All suffered from pains in the legs or feet after walking a few hundred yards, the blood pressure was diminished in the leg most affected (the condition was in each case mainly unilateral) and the arterial pulse was less evident in the affected limb. In one patient the thumb also was the seat of pain and in this man and in the man aged fifty-one pallor of the affected part was noted during the seizure. The patients had all been treated with iodine injections ("Lipiodol"), sodium citrate and diathermy without result. Seven to ten units of "Insulin" were injected before the midday and evening meals every day for twenty days, then ten days' intermission was allowed and the injections were repeated. This procedure was continued for weeks or months according to the relief obtained. At the meals alluded to additional carbohydrates were taken to prevent any hypoglycæmia occurring. The blood sugar of each patient was estimated before treatment commenced and was found to be normal. These four patients obtained relief from their symptoms and the affected arteries became more permeable as a result of this treatment. Other patients with similar disabilities were not relieved and the authors concluded that it was in the so-called juvenile or youthful type of arteritis that this treatment was most useful; in traumatic, infective and senile arteritis little relief was to be expected from "Insulin" treatment.

Paroxysmal Rhinorrhœa.

M. AGAR (*Medical Journal and Record*, September 21, 1927) describes a method of local treatment in hay fever, bronchial asthma and paroxysmal rhinorrhœa. In these conditions part of the skin and mucosa supplied by the fifth cranial nerves is hypersensitive and often itchy. The face should not be touched, rubbed or scratched in any way during an attack. The nasal vestibules should be painted with a mixture consisting of liquid carbolic acid 0·72 mil (twelve minims), oil of peppermint 1·5 mil (twenty-five minims) and rectified spirits of wine to sixty cubic centimetres (two fluid ounces). Then after ten minutes the whole of the outside of the nose, sides of the face, upper lip, lips and chin should be painted. After a few minutes the forehead and scalp should also be painted if they feel irritable. The ears should be examined for cerumen and the outer half of the auditory canal painted. The eyelids should then be painted one at a time and each eye kept tightly closed for about five minutes. A reaction will occur, but the patient should not rub the face or blow the nose, a bowl can be held under the chin. The eyes, face and nose should be blotted with Japanese paper handkerchiefs. When the flow of secretion is exhausted, *lotio calaminæ* warmed should be gently painted all over the face. This treatment should be used two or three times a day or whenever irritation is felt in any area of the face or about the ears. After two days if the patient is not relieved, the nasal vestibules should be painted with silver nitrate one or two grammes to thirty cubic centimetres. Rubbing of these solutions on to the parts is to be avoided, as also is all rubbing of the face, eyelids, nose and ears or scalp.

NEUROLOGY.

Nervous Affections in Tropical Disease.

A. AUSTREGESILO (*Revue Neurologique*, January, 1927), of Rio Janeiro, writes of nervous affections arising in tropical diseases observed in Brazil. First he takes the endemic trypanosomiasis discovered by Chagas, due to the *Schizotrypanum cruzi*, which has an affinity for the nervous system. Children are special sufferers. The acute symptoms are fever, myxœdema, swelling of spleen and liver, followed by phenomena suggesting meningo-encephalitis. In the chronic stage cerebral diplegia is common, at times the paralysis resembles Little's disease, at others pseudobulbar paralysis; also convulsions, athetotic movements and states of mental deficiency may arise. Pathologically accumulations of the *Schizotrypanum cruzi* are to be found in various parts of the nervous system. Treatment must be symptomatic, because those remedies which influence African trypanosomiasis are useless. Next concerning malarial fever, the most frequent accompanying nervous disturbance is coma, which may appear in all degrees; then come meningitic, convulsive and paralytic forms of the disease and even cases resembling acute anterior poliomyelitis and polyneuritis. Beri beri was first recognized in Brazil as far back as the year 1866 and is very prevalent. It occurs in typical form with peripheral neuritis and cardiac disturbance as dominant signs and appears in districts where rice is not consumed. Lack of vitamins in the diet is set down as the determinant. Accompanying ancylostomiasis, it is said that both mental and nervous disturbances of various kinds are very common. They are accounted for by the concurrent grave debility and anæmia. Leprosy, although not a tropical disease, seems to be common in Brazil and, as affecting the nervous system, the neuritic form is the most frequent. Spino-radicular and cerebral forms are exceptional. *Ainhum*, a

disease characterized by slow fibrous strangulation of the toes, occurs in Brazil among imported African negroes and their descendants. It may be a pure trophoneurosis or trauma may play some part. It resembles leprosy, but Hansen's bacillus is absent. Finally, in cases of bubonic plague and yellow fever, over and above nervous manifestations due solely to toxæmia, phenomena of meningitis and encephalitis commonly arise.

Forced Groping and Grasping.

W. J. ADIE and MACDONALD CRITCHLEY (*Brain*, June, 1927) state that, although much has been written on the manifestations of disease in the frontal lobes, the diagnosis of frontal tumour is rarely made with the confidence that sometimes attends localization in other regions, because there are few unequivocal signs of disease confined to this part of the brain. Certain movements, however, of the nature of involuntary grasping and groping which they describe in detail, were observed in three patients with a tumour of one frontal lobe. A search for comparable cases, observed by others, revealed ten like their own and a larger number in which the same signs accompanied vascular lesions. In all these cases the special signs were found in the upper limb of the side opposite to the diseased frontal lobe; in some of the tumour cases they were for a time the only focal signs. The authors are convinced, therefore, that their presence in a patient with a cerebral tumour is unequivocal evidence of the situation of the tumour and the main object of their communication is to draw attention to their value for the practical purpose of localization.

Significance of the Flexor Posture of the Upper Limb in Hemiplegia.

W. RUSSELL BRAIN (*Brain*, June, 1927) shows that the posture of the upper limb in hemiplegia resembles in detail the resting posture of the forelimb which is found in anthropoid apes and monkeys and which is closely related to the assumption by these animals of an erect attitude during rest. Following upon this he indicates that if a hemiplegic human being be made to assume a quadrupedal attitude by leaning forward and supporting the weight of his trunk or as much as he can, on the paralysed arm, such change of posture evokes a reflex extension of the affected limb. This the writer terms the quadrupedal extensor reflex in the upper limb in hemiplegia and he suggests that the posture of hemiplegia is that of the primitive human standing attitude. Moreover, just as Sherrington has shown decerebrate rigidity to be reflex quadrupedal standing, so also the hemiplegic posture may be regarded as reflex erect standing.

Pathology of Achondroplasia.

D. M. ROSSIYSKY (*Revue Neurologique*, February, 1927) describes in detail a case of achondroplasia affecting a man twenty-two years of age and manifesting the typical shortened upper and lower extremities, along with a normal head and body and a normal mind. He also says that although achondroplasia is sometimes inherited, in his case there was no evidence to this effect. Discussing ætiology, he recalls that before the year 1870 the disease was looked upon as a manifestation of inherited syphilis. Then Parrot and others advanced the belief that the affection was purely local, an intrinsic dystrophy of the cartilage of the bones affected. Next the achondroplasic human being was compared with the *Dachshund* and the Basset hound, while a later idea was that he was an atavistic relic of a past race of pigmies. Finally, Marie stated that achondroplasia might be the outcome of endocrinic disturbance. Rossiysky subscribes to this hypothesis and concludes that ætiologically the preponderant part is played by some pituitary disturbance at present obscure.

Sleep, Normal and Pathological.

JEAN LHERMITTE and AUGUSTE TOURNAY (*Revue Neurologique*, June, 1927) were the openers of an important discussion on the subject of normal and pathological sleep, at the annual international reunion of the Neurological Society of Paris held this year. The report is so long that the physiological considerations, interesting though they are, must be omitted in order that the clinical data may be dealt with. First, narcolepsy was taken. This affection remained as originally described by Gelineau and was characterized in its main form by a sudden, irresistible desire to sleep. The attacks are of short duration but of frequent occurrence and if the patient is standing may cause him to fall down. Gelineau thought the condition was a neurosis, so, more recently, did Redlich. The openers of this discussion were opposed to that idea, emphasizing the compelling and wholly irresistible nature of the attack. Actually narcolepsy is the exact opposite to insomnia. Next cataplectic attacks of sleep were considered. These were also known to Gelineau and were characterized intrinsically by a general inhibition of static tone. Suddenly, as the result of emotional disturbance, pleasant or painful, the patient relaxes his muscles and falls asleep. This may last from a few seconds to a few minutes and is not attended by convulsion or loss of consciousness. It was indicated that these attacks are quite different from those of pyknolepsy. In addition to cataplectic attacks, the narcoleptic subject may suffer from ambulatory automatism and likewise experience dreams which almost amount to visions. These three forms were described as illustrating essential or idiopathic narcolepsy. Next a number of forms of symptomatic narcolepsy were described. These, in brief, were the narcolepsy or hypersomnia of cerebral tumour, particularly tumours in the neighbourhood of the third ventricle; the profound somnolence encountered in certain infective diseases of the nervous system, notably syphilis; the narcolepsy following trauma, frequently noticed among troops during the great war; endocrinic syndromes with narcolepsy, for example, the hypersomnia of acromegaly, myxœdema and obesity; the narcolepsies of toxic origin and of epilepsy and hysteria. Next a short chapter is given to continuous hypersomnia as seen in *encephalitis lethargica*, some tumours of the brain and meningitis. The report ends with an inconclusive discussion on the localization of a sleep "centre" in the brain.

Herpes Zoster.

G. MARINESCO and ST. DRAGANESCO (*La Presse Médicale*, August 20, 1927) advance a new view on the pathology of *herpes zoster*. They maintain that the cutaneous or mucous eruptions characterizing this disease are the result of the action of the specific virus directly upon the skin; the eruptions do not represent trophic disturbance consequent upon lesion of the posterior root ganglion, which has been the accepted doctrine; the lesions found in posterior root ganglia and the accompanying changes in spinal cord and nerve roots form part of an ascending neuritis, the primary infection having been in the skin and propagated therefrom along the nerve. In support of their view they report first a case of *herpes zoster* affecting the thigh of a hemiplegic patient, paralysed on the same side, who at death four years later manifested chronic inflammatory changes in the right first lumbar posterior root ganglion and in the originally affected skin. They emphasize the point that the eruption appeared on skin primarily affected by a thalamic hemiplegia-producing lesion. Secondly, they point to the number of instances in which irritative or destructive lesions of posterior roots and Gasserian ganglia have arisen and been unaccompanied by *herpes zoster*. Finally, they admit that the possibility of a spread of infection in the reverse direction, namely from ganglion to skin, cannot be wholly denied.

Treatment of General Paralysis with "Stovarsol."

A. SÉZARY and A. BARBÉ (*Revue Neurologique*, June, 1927), after treating a series of sixty-eight patients suffering from general paralysis of the insane with "Stovarsol" claim results equal in benefit to those obtainable with malarial therapy. They recommend a course of twenty-one intravenous injections spread over a period of seven weeks. The drug should be dissolved in twelve cubic centimetres of distilled water, half a gramme for the first and one gramme for each subsequent dose. If these doses be not exceeded, optic neuritis, a recognized danger, may be averted.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION TOGETHER WITH THE SECTION OF MEDICINE AND THE SECTION OF PATHOLOGY AND BACTERIOLOGY was held at the B.M.A. Building, 30 to 34, Elizabeth Street, Sydney, DR. S. A. SMITH, the President, in the chair.

The Anæmias.

DR. A. W. HOLMES À COURT read a paper entitled "The Anæmias; the Medical Aspect" (see page 7).

DR. ELSIE DALYELL read a paper by herself and DR. MARIE M. HAMILTON entitled: "A Series of Blood Examinations Made in a Public Health Laboratory During a Period of Three Years" and illustrated her remarks with lantern slide pictures.

They presented an analysis of all blood examinations carried out at the Board of Health over a period of three and a half years.

Reports on many specimens received from country practitioners were made on microscopic examinations of stained films alone and full blood examinations were made in comparatively few instances. The information available was, therefore, incomplete, but as confirmatory examinations were made where necessary, the diagnoses could usually be established with reasonable accuracy.

The total number of specimens examined was 1,005. No abnormality was noted in 322. Differential counts and examinations for parasites were carried out 142 times. Secondary anæmia was diagnosed 398 times, chlorosis nine times, definite pernicious anæmia 89 times, probable pernicious anæmia five times and leuchæmia 21 times. In eleven instances the blood of adults and in eight the blood of children could not be classified.

Of the eleven adults whose condition was unclassified, five were suffering from severe anæmias complicating pregnancy and showing the characteristic picture of pernicious anæmia.

The unclassified conditions in infancy included four cases with features of von Jaksch's anæmia and four cases of severe secondary anæmia with indications of hæmorrhagic diatheses, but in each instance the hæmorrhage was of obscure origin.

The proportion of cases of pernicious anæmia appeared high, but it was not possible to obtain exact figures regarding the relative incidence of this condition. Vital statistics did not differentiate between primary and secondary anæmia, so that the figures for New South Wales could not be compared with figures quoted from other areas. The reports from Royal Prince Alfred Hospital and Sydney Hospital revealed 191 cases of pernicious anæmia treated at these hospitals in three years. Recent studies of hospital records showed that 117 cases were treated at London Hospital in ten years and 111 cases in five years at Johns Hopkins Hospital, but it is uncertain whether these figures were comparable with those of the Sydney hospitals.

The most interesting cases were those of severe anæmia associated with pregnancy which were often indistinguishable from pernicious anæmia, although recovery occurred on termination of the pregnancy. Recent publications by Larrabee and Panton and his colleagues devoted much attention to this question and emphasized the need for caution in diagnosis of pernicious anæmia in puerperal woman.

DR. F. GUY GRIFFITHS expressed the opinion that Dr. Holmes à Court's interesting and suggestive summary would form an excellent basis for discussion. Dr. Dalyell and Dr. Hamilton's contribution from the pathological side should prove valuable to all medical men and women in actual practice. In regard to the word pernicious he was not quite in agreement with Dr. Holmes à Court. Pernicious did not necessarily imply mortal. There were many pernicious things that they would be glad to eradicate and could not. He had seen many patients with pernicious anæmia live for years after the disease had manifested itself. He doubted whether it would be possible to discontinue the use of the term after it had been in use for so long. Turning to the question of the treatment introduced by Minot and Murphy he stated that it was interesting to learn that one hundred and fifty grammes of liver at times induced nausea. His experience was that patients refused liver after a week and that the dislike became increasingly pronounced. He was satisfied that arsenic when given by the mouth was the most useful remedy for pernicious anæmia. It could be rendered more valuable by the addition of hydrochloric acid. There was no objection to the combination; this could be carried out by giving small doses of *liquor arsenici hydrochloricus* (four to six drops) together with increasing doses of hydrochloric acid.

He expressed his sympathy with Dr. Dalyell that she had not been able to obtain fresh specimens of blood. He thought that the immediate examination of a drop of fresh blood yielded extremely valuable information. Although a differentiation between the forms of leucocytes could not be made, the examination of fresh, unstained preparations enabled one to form a reliable opinion and sometimes even a diagnosis of pernicious anæmia or of malaria. He recalled the fact that Laveran had discovered the plasmodium of malaria in unstained, fresh specimens of blood. The shape of the red corpuscles and their actual size could be gauged better in fresh blood than in smeared films.

Reverting to the medication with arsenic and hydrochloric acid, he said that he was inclined to think that arsenic was the best single drug. He had recently treated three patients with arsenic and hydrochloric acid combined, a second, whose anæmia was more severe than the first, had been given arsenic without other medication; a third had been treated with arsenic, then with transfusion of blood and later with arsenic, hydrochloric acid and liver. The second patient had responded quite as well as the other two and had remained well for some months.

He referred to Dr. Dalyell's case for diagnosis and suggested that it might have been a lymphosarcoma with metastasis in the bone marrow. This would account for the presence of myeloblasts. In conclusion Dr. Griffiths spoke of the condition that Dr. Dalyell had found rare in Australia, namely von Jaksch's anæmia. The late W. F. Litchfield had described a case of this disease in the early years of the war.[1] Dr. Griffiths suggested that it was sometimes overlooked.

DR. A. R. COLWELL gave some details of a case of pernicious anæmia of six years' standing. A man, aged fifty-two years, had suffered from an attack of influenza in July, 1922. He had returned to his work after a week, but had been unable to continue. Dr. A. E. Mills had diagnosed pernicious anæmia. In August, 1923, he had been treated with blood transfusion. His anæmia had been severe; there were 2,180,000 red blood corpuscles per cubic millimetre of blood and 5,000 leucocytes. Transfusion had been repeated at long intervals. Relapses had occurred at Christmas time, 1926, but in February of 1927 he had worked for a while. His health had improved without treatment up to September, 1927. At that time there were 2,400,000 red blood corpuscles per cubic millimetre and 12,800 white corpuscles. The anæmia, therefore, had remained stationary. In September he was given 250 grammes of liver, 300 grammes of brain and 300 grammes of kidney daily. After three weeks his red blood corpuscle figure had increased to 3,200,000 and the white cell count was 9,400. A further improvement had been registered in the following three weeks and at the end of nine weeks' treatment his red cell count was 4,300,000 and white cell count 9,200. The cells were practically normal. Two weeks after the commencement of the treatment he had walked half a mile without being

[1] On May 25, 1917, Dr. Burton Bradley reported a case of severe anæmia of the pernicious type in an infant and after considering three possibilities, namely true pernicious anæmia in an infant, secondary anæmia from some undiscovered cause and Somma's *anæmia splenica* otherwise known as von Jaksch's *anæmia infantum pseudoleukæmica*, he left the diagnosis open. The late W. F. Litchfield discussed von Jaksch's anæmia of infants and came to the conclusion that Dr. Bradley's patient had a secondary anæmia. Dr. Guy Griffiths also took part in the discussion.—EDITOR.

fatigued. After twelve weeks' treatment his red cells numbered 4,750,000 and leucocytes 8,100. The red cells were normal. He looked well and was robust and active. The only treatment he had had consisted of liver, brain and kidney in his diet.

DR. O. A. A. DIETHELM referred to a patient he had seen in June. The blood examination had revealed that there were 2,020,000 red corpuscles per cubic millimetre, 4,500 white corpuscles and the colour index was 1·2 with 52% hæmoglobin. His blood contained megaloblasts and myelocytes. On August 3 he was given Minot and Murphy's diet and hydrochloric acid up to 3·5 cubic centimetres. On August 6 his red blood corpuscles had increased in number to 4,200,000 per cubic millimetre and the colour index was 1·1. The blood picture had improved very considerably. In October he was looking well, not in the least like a patient with pernicious anæmia. His blood was practically normal. He was taking the diet well. His daily ration of liver was between 120 and 140 grammes of liver with raw beef and so forth. In reference to Dr. Dalyell's patient with splenomegaly he stated that he had seen a patient with a similar condition. The colour index was high; there was definite achylia, but at no time was there any interference with the functions of the leucocytes. He had come to the conclusion that the condition was pernicious anæmia.

DR. A. H. TEBBUTT, D.S.O., rose to take part in the discussion chiefly to encourage the pathologists to have their say. He was inclined to be careful in regard to the whole subject of the primary anæmias, for it seemed as if they might have to eat their words within a short time if they ventured on definite opinions at the present time. He wished to join issue with Dr. Guy Griffiths in regard to the examination of fresh blood specimens in diagnosis. No one placed any reliance on unstained specimens alone. The pathologist's task in connexion with the anæmias was a complex and difficult one. Greater thoroughness was necessary and they should be careful not to revert to the methods in vogue when Lavaran carried out his malaria work, but rather to try new methods of staining. In regard to the use of the word pernicious it must be remembered that some patients appeared to recover or at any rate lived for many years. The diagnosis was not permissible until a full hæmatological examination had been carried out. He referred to the caution needed in estimating the value of treatment. He remembered a patient who had had the disease for eight or nine years. He had contracted the disease before the advent of treatment by large doses of hydrochloric acid or liver feeding. The patient was still holding a responsible position and was still able to perform arduous duties. They spoke of Addison's anæmia. The Germans had a way of ignoring the priority of Englishmen who described diseases. They called Addisonian anæmia Biermer's anæmia; they called Graves's disease *morbus Basedowii*. The ætiology of pernicious anæmia was still undecided. It was difficult to reconcile Hurst's modification of Hunter's hypothesis of oral sepsis with the fact that some patients had good remissions with liver feeding alone. Again he called attention to the occurrence of typical chlorotic anæmia in women between the ages of twenty-five and fifty. The textbooks would lead the clinician to refuse the diagnosis in any patients other than girls and young women. The anæmia was idiopathic; there had been no hæmorrhage and no discoverable sepsis.

He had examined the records of the Royal Prince Alfred Hospital of the past ten years and had found that fourteen patients had been treated for lymphatic leuchæmia and thirty for myeloid leucæmia. The mortality among the former had been 54% and among the latter 40%. He recognized that this meant that the patients who did not die in hospital were able to leave the hospital, but may have died elsewhere.

No cases had been diagnosed as acute myeloblastic leuchæmia, though textbooks stated that this was not an uncommon form. It was probable that the physicians had placed them in the lymphatic group. The pathologists were responsible for the error. It was often very difficult to distinguish myeloblasts from lymphoblasts and other cells might be present in small numbers. Lymphatic leuchæmia occurred in patients who were younger than those affected with myeloid leuchæmia. All the patients of the first group were males. The number of white cells was lower in the lymphatic form than in the myeloid. In acute leuchæmia there was some fever; in chronic leuchæmia there was little or none. In conclusion Dr. Tebbutt claimed that pathologists should take an interest in blood work. New methods of staining and examination of blood were being introduced and it was not possible to determine which was the best or which was essential.

DR. L. UTZ held that the science of hæmatology was in a somewhat chaotic state. A struggle was taking place between the supporters of the monophyletic and those of the polyphyletic theories of the cell. He referred to Professor Witherington Stump's valuable article read at the Dunedin session of the Australasian Medical Congress and published in THE MEDICAL JOURNAL OF AUSTRALIA of November 19, 1927. There was much confusion in the study of hæmatology owing to the varied nomenclature. For example there are no less than eight hundred synonyms for a single cell. In considering the evolution of the complete blood picture it was necessary to realize that there was a gradual merging of one type into another. It was common to name every intermediate stage in this transition, but since the stages were often ill defined, the introduction of separate names led to great confusion. He thought that the greatest caution should be exercised in regard to the hypotheses of the ætiology of pernicious anæmia. The recent discussion in England at which Gulland, Hurst and many other authorities had spoken, illustrated this need. Piney held the view that the presence of embryonic rests was essential for the production of pernicious anæmia. Everyone admitted that there was a distinct hereditary tendency toward the disease and that there was an associated achlorhydria. But no one had been able to account for the association of achlorhydria and pernicious anæmia. It seemed to be a precursor, not a cause nor an effect.

DR. E. MARJORIE LITTLE said that the members of the Section of Pathology and Bacteriology proposed to work at some of the problems raised in recent times in connexion with hæmatology. Pathologists agreed in regard to the diagnosis of pernicious anæmia, but disagreed on many of the finer points. She agreed with Dr. Utz that there was a deplorable confusion of terms.

DR. C. H. SHEARMAN supported Dr. Tebbutt in his objection to the use of fresh, unstained specimens of blood. Much more information could be gained from a carefully made smear, properly stained. A leucopenia associated with a colour index above unity was of great value in arriving at a diagnosis, but frequently the stained blood smear gave information which enabled the diagnosis to be definitely settled. The leucopenia was not always present. Dr. Shearman referred to two patients seen recently in whose blood the changes in the red cells as seen in smears with a colour index above unity and an associated achlorhydria left no doubt as to the diagnosis; yet in each a persistent leucocytosis was present. Further errors in the estimation of the hæmoglobin value resulted in faulty estimation of the colour index. Dr. Shearman found that the standard methods of measuring hæmoglobin were in need of review. Variations of as much as 10% were not uncommon when different methods of estimation were employed. Such a range of error might easily result in colour indices below unity being obtained in cases of Addisonian anæmia or above unity in cases of secondary anæmia. In these circumstances Dr. Shearman was of the opinion that the stained smear was of the greatest value in enabling the hæmatologist to arrive at a diagnosis.

In conclusion Dr. Shearman stated that the methods of staining blood films needed revision, if they were to follow the teaching of Piney.

DR. M. S. VEECH pointed out that Hurst's observations had revealed that the absence of free hydrochloric acid predisposed to pernicious anæmia. It was known that 90% of people with achlorhydria ultimately suffered from this disease. This was really the extent of their knowledge of the ætiology of the disease. In regard to liver treatment he warned against the use of the liver extracts at present on the market. Macleod claimed that he had an extract that was reliable. He, the speaker, had no direct information about it. In *The British Medical Journal* of October 15, 1927, there was an article by Hurst in which

the author gave an admirable account of the present state of their knowledge. He maintained that the best treatment was still with arsenic and iron. If the colour index was lower than one, iron was indicated. Otherwise it was advisable to give arsenic.

DR. S. A. SMITH, the President, in closing the discussion referred to the debates that had taken place in Edinburgh and in America within the past year. There appeared to be a wave of optimism in the air about the disease. If one listened to the exponents of the various ætiological views, one was convinced successively of their accuracy. The belief that pernicious anæmia was a toxæmic process was widely held and many claimed that the toxin was a product of the bacillus of Welch. Many English authorities were convinced that in spite of all new forms of treatment, arsenic was the great standby. The claims of the Boston school were not supported in most parts of the United States of America, but the 100% claims of Minot and Murphy were not accepted everywhere. In regard to this treatment the palatability of liver constituted a grave difficulty. All sorts of preparations of cooked liver were being tried. While he was in the United States he had learned that they had even invented a liver cocktail. He had not tasted it. It was said to be as pleasant as any other cocktail in the States. Judging from those he had tasted in America, he would conclude that this was no high praise. Since his return he had attempted to give liver, with somewhat disastrous results in some instances. Nausea had followed even if less than two hundred grammes of liver were given. Good results were obtained if the liver were combined with tomatoes in sandwiches. The Carnrick Company was, he understood, preparing an extract of liver in concentrated form that was reasonably palatable and that could be taken in fairly large quantities over a considerable period. The British clinicians were still sceptical. This was not altogether surprising, since the method had been known for only a few months. It had been found that some patients would not take the large liver ration suggested.

In regard to Dr. Dalyell's patient who manifested signs of anæmia with megaloblasts in the blood during the puerperium, he spoke of a patient who had had a similar condition after labour and *post partum* hæmorrhage. She had been treated by transfusion and hydrochloric acid on account of her achlorhydria. She recovered from the first attack. At her second pregnancy she suffered a relapse which was also followed by recovery. He did not know whether the patient would recover from a second relapse. He remembered another patient who had been under treatment over six years previously. Recovery had taken place and there had been no relapse, but the patient had not again become pregnant. In connexion with anæmia with splenomegaly he remembered a patient in the Royal Prince Alfred Hospital, whose blood picture justified the diagnosis of Addisonian anæmia. At first there had been no splenomegaly, but the clinical picture had been difficult to explain. The enlargement of the spleen had developed later. He thanked the authors for their stimulating papers.

Before Dr. Holmes à Court rose to reply, Dr. F. Guy Griffiths asked to be allowed to explain that he had not intended to convey the impression that he preferred to rely on fresh blood specimens rather than stained smears. He claimed that one could obtain a reliable general impression on which a provisional diagnosis could be made.

DR. HOLMES À COURT thanked those who had taken part in the discussion for the interesting remarks they had made. He agreed with Dr. Tebbutt in regard to the dangers of dogmatism on this half understood subject. They had advanced in their manangement of the disease. He congratulated Dr. Dalyell on her excellent demonstration of the slides. Smears sent by practitioners in the country were often bad. He thought that the prognosis in pernicious anæmia was usually good. On the other hand he remembered one patient who had been treated with transfusion and had died within twenty-four hours, probably of sepsis. He suggested that the condition from which Dr. Dalyell's patient with leucosarcoma and pernicious anæmia was suffering might have been Sternberg's disease. He had seen only two children with von Jaksch's disease. It was probably a deficiency disease. He advocated a suspension of judgement in regard to treatment at the time. The future would reveal something of value.

DR. DALYELL had been interested to hear of the various methods of disguising liver. She had heard it suggested that liver with ice cream was quite palatable. In regard to the matter of recovery from true pernicious anæmia, she reminded her audience that Panton had found that among 117 patients with pernicious anæmia in the course of ten years, only eight had been alive at the end of that time. She thought that Dr. Tebbutt had been a little unkind about the Germans and Austrians. They always called rickets *die englische Krankheit* (the English disease) as if the English had been responsible for the occurrence of this condition. Again they called the infantile form of osteomyelitis Barlow, after Sir Thomas Barlow, who had described it. They pronounced the second syllable "off." She thought that the reason why they were seeing more cases of von Jaksch's disease was the extension of industrialism. It was probably of alimentary origin. The association of purpura and leucocytosis was fairly definite. It was a primary condition, but needed much further investigation. The whole nomenclature and classification of the blood elements should be revised.

MEDICO-POLITICAL.

AT a meeting of the Eastern Suburbs "Division" of the Victorian Branch of the British Medical Association held recently it was resolved:

> That this meeting is of opinion that the qualification F.C.S.A. as at present constituted is inimical to the best interests of the profession and the community.

Correspondence.

THE HOSPITAL PROBLEM.

SIR: The very clear exposition of the hospital problem of Melbourne and the Commonwealth placed before readers of THE MEDICAL JOURNAL OF AUSTRALIA by Dr. D. M. Embleton merits the serious attention of the medical profession and the public, especially those of its members entrusted with the task of government.

There are several remarks of a general nature in Dr. Embleton's paper that call for comment. "Any solution which does not adequately reward and thereby cultivate the skilful employment of the highest ability in the maintenance of health is an unsatisfactory solution." An adequate reward is governed by economic necessity and trite as this observation may appear at first sight, it is the crux of the whole problem.

"The solution of the problem is evolving without any collective professional sanction." Are we not as a profession unwise to allow this state of affairs to continue and while such criticism applies if the solution of the problem is not to our liking, are we not ourselves to blame?

It seems obvious to the writer that from the time of the Harvester case onwards right up to the reign of Mr. Piddington the medical profession has failed to see (or been indifferent to) signs and portents that should have been obvious.

Dr. Embleton says Mr. Piddington deals lightly with the subject of the provision of nursing and medical attention in connexion with "awards."

Perhaps one should not blame Mr. Piddington for having appeared to deal lightly with the subject as he may have assumed that those members of the medical profession engaged in friendly society practice were quite satisfied with the capitation fee received from members of the friendly societies.

It is most difficult to ascertain what factors were taken into consideration in fixing the capitation fee in the early days of friendly society practice.

If one asks the question why is the capitation fee in any State a particular amount, a satisfactory answer is

not forthcoming. As the cost of living and the rate of wage does not vary greatly throughout the Commonwealth and at any rate the differences are not great in the capital cities, one might reasonaly expect that the capitation fee would be the same all over Australia.

If there is to be any variation in the capitation fee in the Federal Model Lodge Agreement, would it not be wise to determine the variation according to the cost of living and rate of wages in the various parts.

It was most unfortunate that time did not permit of this subject being fully discussed by the conference representing members holding lodge appointments that met in Melbourne in April, 1926.

Now it would seem reasonable that the amount of the capitation fee for friendly society practice should be determined by the economic status of the members of the friendly society and the nature of the service rendered by those members of the medical profession contracting to do this class of work.

So far as one can determine, nobody has scientifically attempted to determine how much friendly societies can pay for services rendered by the medical profession. Up to the present the main idea seems to have been to obtain these services as cheaply as possible.

Should the friendly societies be unable to pay a fair remuneration for services rendered, the medical profession perforce have to modify their demands. This is where the idea of friendly society practice savouring of charity arises.

This brings one to a consideration of the position where friendly society practice has a bearing on the hospital problem.

Last year certain friendly society members approached the Queensland Branch of the British Medical Association with a request to consider the possibility of providing hospital treatment. The gentlemen in question stated: "The insurance of our members against hospital treatment in addition to medical attendance, medicine, sickness and funeral benefits will place the coping stone on friendly society activities."

It was proposed to provide for hospital maintenance by a levy of threepence per week per member desiring insurance.

The question of providing hospital attendance was considered, but there are certain administrative difficulties and other difficulties, for example, the fact that certain of the medical attendants lived a long distance from the hospital.

The whole scheme is still *sub judice.*

It seems clear, however, that should the Federal Model Lodge Agreement come into force in Queensland, it will be obligatory for practitioners holding lodge appointments to include hospital attention as part of the service rendered.

As taxpayers we should be glad to see the friendly society members paying for their hospital maintenance, but even so members of the profession engaged in contract practice will be well advised to try to obtain a reasonable capitation fee.

Dr. Embleton quotes our Mr. Stopford (who is always most considerate to the profession in Queensland) who expressed the altered popular attitude to hospitals thus: "Hospitals are regarded in Queensland as services of equal importance to water reticulation and sewerage and not as charitable institutions to the poor."

It must be remembered, however, that all the workers in the latter services are paid and while not wishing to enter into the pros or cons of the question of the abolition of the honorary system, one is of the opinion that we must do our best at all times to urge the authorities to take the utmost care to see that there is no exploitation of the honorary system by those able to pay.

The hospital problem is with us and the longer we delay to deal satisfactorily with it, the more complex it will become.

Its solution demands the attention of the members of our profession and of all those people it affects. It should be needless to remark that all those attempting to solve the problem should work amicably together. We could do well to emulate the parent Association in this respect. Each word of the Right Honourable Neville Chamberlain at the last autumn dinner of the parent Association speaks volumes for the harmonious relations existing between the British Ministry of Health and the medical profession.

The Federal Committee might entertain the Commonwealth and State Ministers of Health when next they meet.

Would it not be possible for the Federal Committee to request each Branch and each local association in Australia to give immediate consideration to the hospital problem, thus in the words of Dr. Embleton offering the whole profession the opportunity to coordinate its outlook and requirements and negotiate with hospital and public in accordance therewith?

Yours, etc.,
E. S. MEYERS, M.B. (Syd.),
Honorary Surgeon, Brisbane Hospital.

Brisbane.
December 7, 1927.

Post-Graduate Work.

POST-GRADUATE COURSES IN BERLIN.

INTERNATIONAL post-graduate courses in medicine will be held in Berlin by the Union of Professors and by the organization centred in the Kaiserin Freidrich Institute with the assistance of the medical faculty of the University of Berlin. One section of the courses is held continuously and another section will be held during March and April, 1928.

The permanent courses are of two kinds. One series lasts from two to four weeks and another is thrown open to those who wish to attend lectures in clinics, hospitals and laboratories. These last for two to three months or possibly longer and are intended for those who wish to gain practical experience from systematic instruction.

The special courses in March and April, 1928, will cover several subjects. One course, to last a fortnight, will be concerned with "new aspects and methods of clinical pathology and therapeutics." Another course which will last ten days, will deal with diseases of metabolism. A third course also to last ten days, will be devoted to obstetrics and gynæcology. From April 17 to 24, 1928, special consideration will be given to "improvement in Röntgen diagnosis and ray therapeutics covering the whole medical ground." Other courses of certain lecturers will cover the whole range of medicine; these courses are combined with practical work. Instruction is given in German, but numerous professors are able to lecture in English. Participants in these courses after due registration receive a certificate if they desire it. The office quarters are situated in Kaiserin Freidrich Haus, Berlin, N.W. 6, Luisenplatz 2-4. Further information may be obtained from the office and assistance will be given in finding suitable lodgings and in arranging visits to various clinics.

Proceedings of the Australian Medical Boards.

NEW SOUTH WALES.

THE undermentioned have been registered under the provisions of *The Medical Act*, 1912 *and* 1915, of New South Wales, as duly qualified medical practitioners:

Brentnall, Harry Austin, L.R.C.P. (Edinburgh), 1927; L.R.C.S. (Edinburgh), 1927; L.R.F.P.S. (Glasgow), 1927; Queanbeyan.

Bush, Frederick Keith, M.B., B.S., 1926 (Univ. Melbourne); Moulamein.

Eppel, David, L.L.M.R.C.P. *et* S. (Ireland), 1922; c.o. Commercial Banking Company, Sydney.

Plummer, Beryl Kent, M.B., B.S., 1927 (Univ. Melbourne); District Hospital, Wagga.
Rawlings, William Joseph, M.B., B.S., 1927 (Univ. Melbourne); District Hospital, Albury.
Rivett, Edward William, M.B., 1920; Ch.M., 1921 (Univ. Sydney).

For additional registration:

Chesterman, John Nicholson, F.R.C.S. (Edinburgh), 1927.

TASMANIA.

The undermentioned have been registered under the provisions of *The Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

Fox, Raymond William Sydney, M.B., B.S., 1927 (Univ. Melbourne); Alfred Hospital, Melbourne.
Renou, Cecil Ashley Marshal, M.B., B.S., 1925 (Univ. Melbourne); Gormanston.

Books Received.

SYNOPSIS OF PHYSIOLOGY, by A. Rendle Short, M.D., B.S., B.Sc., F.R.C.S., and C. I. Ham, M.B., B.Ch., M.R.C.S., L.R.C.P.; 1927. Bristol: John Wright and Sons, Limited. Crown 8vo., pp. 258, with illustrations. Price: 10s. 6d. net.

A MANUAL OF CHEMISTRY, by W. Simon, Ph.D., M.D., and Daniel Base, Ph.D.; Thirteenth Edition, Enlarged and Thoroughly Revised by John C. Krantz, Jr., Phar.B., Sc.D.; 1927. London: Baillière, Tindall and Cox. Royal 8vo., pp. 711, with illustrations. Price: 25s. net.

Diary for the Month.

JAN. 10.—Tasmanian Branch, B.M.A.: Branch.
JAN. 10.—New South Wales Branch, B.M.A.: Council.
JAN. 13.—Queensland Branch, B.M.A.: Council.
JAN. 16.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JAN. 17.—Tasmanian Branch, B.M.A.: Council.
JAN. 17.—New South Wales Branch, B.M.A.: Ethics Committee; Executive and Finance Committee.
JAN. 18.—Western Medical Association, New South Wales.
JAN. 19.—Victorian Branch, B.M.A.: Council.
JAN. 24.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JAN. 27.—Queensland Branch, B.M.A.: Council.

Medical Appointments.

Dr. Lorimer Dods (B.M.A.), Dr. D. G. R. Vickery (B.M.A.) and Dr. C. E. Winston (B.M.A.) have been appointed Temporary Honorary Relieving Medical Officers to the Royal Alexandra Hospital for Children, Sydney.

* * * *

Dr. Thomas Lynewolde Anderson (B.M.A.), Dr. Herbert John Gray (B.M.A.) and Dr. Joseph Patrick Kenny (B.M.A.) have been appointed to constitute the Dental Board of Western Australia for the years 1928, 1929 and 1930.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xx.

AUSTIN HOSPITAL FOR CHRONIC DISEASES, HEIDELBERG, VICTORIA: Resident Radiologist and Pathologist.
AUSTRALIAN INLAND MISSION: Flying Doctor for Cloncurry.
ST. VINCENT'S HOSPITAL, SYDNEY: Honorary Gynæcologist, Honorary Assistant Gynæcologist.
SYDNEY HOSPITAL: Clinical Assistant.
THE ADELAIDE CHILDREN'S HOSPITAL, INCORPORATED: Three Resident Medical Officers.
THE UNIVERSITY OF MELBOURNE: Director of Bacteriological Laboratory.
THE UNIVERSITY OF SYDNEY: Radiotheropeutist to the Cancer Research Committee.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Secretary, 267, North Terrace, Adelaide.	All Contract Practice Appointments in South Australia. Booleroo Centre Medical Club.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to position at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

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